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MITTELSCHMERZ OR OVULATION PAIN *

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The purpose of this paper is to draw the attention of the profession to the fact that large numbers of young women are being operated on unnecessarily for appendicitis with mutilation of a healthy ovary, when the true diagnosis is ovulation pain or 'Mittelschmerz.'

Incidence of Ovulation Pain. It is estimated that between 15% and 40% of women suffer from this condition (McSweeney and Fallon, 1950). It occurs in these women almost monthly and therefore is much commoner than the common cold. There is no information available regarding the incidence in Bantu women, but my experience in out-patient clinics is that Bantu women rarely complain of it—possibly because they are more stoical.

Symptoms. The attacks take place usually at the mid-cycle, but may occur any time between the 7th and 24th days of the cycle. They do not necessarily occur every month. These pains, like dysmenorrhoea, are not as a rule experienced until a few years after the menarche, i.e. until true ovulatory cycles have been established. The young woman is then surprised by a pain which may vary between a mild ache and an agonising, shocking pain. It is present in one or other iliac fossa, usually the right. In some cases a mild backache may be present. The pain is most often bearable but the patient and her parents are worried about the danger of appendicitis, of which condition they have heard on countless occasions; the much commoner mittelschmerz has not been publicized. Usually the pain subsides within a few hours, but in some patients, especially those who have suffered meddlesome surgery to the ovary, the pain may continue until menstruation begins. It was this type of patient that O'Donel Browne (1949, 1950) had in mind when he described 'ovarian dysmenorrhoea'.

A few patients, relatively rare, bleed freely from the corpus luteum at the time of ovulation and suffer generalized abdominal pain and distension with a raised temperature. There must be few gynaecologists who have not, at some time, diagnosed such a case as an ectopic pregnancy. Some patients may present with such symptoms month after month. This condition should be regarded as a separate clinical entity, viz. corpus luteum haemorrhage, rather than true mittelschmerz.

Aetiology. The exact mechanism of the production of mittelschmerz has not been established. Of various theories the most likely is that there is raised intra-ovarian tension immediately before ovulation. The fact that similar pain can be elicited by bimanually compressing the ovary strongly supports this view.

Diagnosis.

The patient herself unfortunately seldom recognizes the fact that her pain is related to the menstrual cycle. Where the pain is on the right (the usual side) she generally suspects appendicitis. This diagnosis is suggested to her doctor, who usually agrees. He has been taught to be on his guard against appendicitis, whereas he has scarcely heard of ovulation pains; he can therefore hardly be blamed.

On the other hand, the practitioner (all too rare at present) who appreciates the probabilities involved will be at pains to exclude the common mittelschmerz before considering the proportionately rare appendicitis. The possibility that the pain might occur every month at mid-cycle has to be suggested to the patient. Even then many will say that they have never noticed and cannot say. The next enquiry to make is whether the pain ever occurs during menstruation. On this point sufferers from mittelschmerz can always definitely answer 'No'. Such a reply is almost diagnostic, since other lower abdominal pains are either worse during menstruation or are unaffected by it. The diagnosis is then confirmed by bimanually compressing the ovary, which reproduces the pain of which the patient complains.

TREATMENT

It is almost a tradition in operative surgery that incisions for appendicitis in women should be planned in order to deal with alternative adnexal pathology. In endless numbers of cases this procedure is adopted for mittelschmerz. A

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normal appendix is removed and the 'pathology' in the ovary (a corpus luteum, normal follicle or harmless retention cyst) is dealt with by taking a wedge out of the ovary, or even completely removing it. It is a fact which I have confirmed from the operation register of a Reef hospital that in some quarters the commonest operation in young women is appendicectomy with removal of a portion of an ovary. In the 14-25 age-group appendicectomy at this hospital was more than twice as common in women as it was in men. In the minority of cases, where the ovulation pain is left-sided, the patient is less likely to suffer unnecessary mutilation.

An astonishing feature of the post-operative history is that the patient does not blame the doctor when the pain recurs often in a more persistent form. Such is the power of suggestion associated with any operation that the patient now thinks it is a new pain which afflicts her—possibly cancer! 'Surgery begets surgery' is an old saying, which should read, 'Bad surgery begets surgery'. Now our patient is liable to have operations for adhesions or for more cysts, or a ventrisuspension or removal of an ovary, etc. and eventually hysterectomy!

The following is the history of a badly mismanaged case —admittedly more disastrous than most, but none the less typical.

The patient, a married woman aged 35 without children, had started complaining of right-sided pains at the age of 17. These on careful consideration of her history, were obviously ovulation pains, but they were never diagnosed as such. Instead, an appendicectomy was performed, with removal of a 'cyst'.

When pains recurred an operation for adhesions was carried out. This was followed over the next 10 years by operations for further cysts, cholecystectomy, ventrisuspension, and finally hysterectomy—but leaving the patient with her ovaries. Unfortunately the pains continued, rather irregularly now, but more severely.

The urologists were next called in and carried out full investigations, which proved negative. The patient was then referred to a psychiatrist, who gave her shock therapy, with negative results. In despair the patient was referred to a gynaecologist, to whom it was clear that the pain had always been an ovulation pain.

It was decided to remove the offending right ovary, which could be felt on pelvic examination as a tender cystic mass the size of a golf ball. At laparotomy such dense adhesions were found that it was feared that the closely adjacent ureter or bowel might be injured, and so the ovarian vessels with their nervous accompaniments were divided (as will be discussed presently) and the ovary was left amongst its dense adhesions. The patient unfortunately developed post-operative intestinal obstruction, for which she had her abdomen reopened by a general surgeon. She made a slow and stormy recovery, with final disappearance of her ovulation pain. Subsequently she developed an incisional hernia which necessitated yet another and (one hopes) a final operation.

Once the correct diagnosis has been made the treatment is obvious and easy. The true nature of the pain is explained to the patient, who is then quite willing to tolerate even quite severe pain with the help of an analgesic if necessary. She is told the pain will finally disappear with the menopause and that it is a sign that normal ovulation is occurring.

In a very small minority of cases, especially where the pain has become unbearable after surgical mishandling, or where there is doubt whether endometriosis etc. may be present, a laparotomy may be necessary. If no genuine ovarian pathology is present an ovarian neurectomy should be performed (O'Donel Browne, 1949, 1950; Henrickson, 1941). This entails dividing the sympathetic and parasympathetic nerves in the infundibulo-pelvic ligament. The only practical way of doing this is to divide the whole ligament, including the ovarian vessels. This does not interfere with subsequent ovarian function because the uterine vessels which anastomose with the ovarian are intact. As it is possible that some of the sympathetic and parasympathetic nerves may ascend by way of the presacral plexus, it is advisable to do a presacral neurectomy at the same time.

In all, 7 cases have been treated by the author by ovarian sympathectomy, 3 of them in combination with presacral neurectomy. The results have been uniformly good.

DISCUSSION

Why is such a well-recognized condition as mittelschmerz pain, of which the attacks are much more prevalent than the common cold, so poorly handled by our profession? The fault lies primarily with the medical schools, where the condition has never been given the emphasis it deserves. All competent surgeons and gynaecologists are aware of the condition but even with them it is not always uppermost in the mind when a young woman complains of obscure abdominal pains. Many well-known text-books on gynaecology and surgery do not even mention the condition. It should be drummed into all students that in lower abdominal pain in a young woman the diagnosis should never be made without first carefully excluding ovulation pain. In this way thousands of unnecessary and mutilating operations could be avoided. It would also be an excellent thing if writers on medical matters in the lay press and speakers on the radio were to give this ubiquitous condition their attention.

SUMMARY

Mittelschmerz or ovulation pain occurs in approximately one-third of all women. Its attacks are therefore much commoner than the common cold. It is nevertheless misdiagnosed and mishandled in a high proportion of cases, often with disastrous results. The fault lies primarily with the medical schools, where students should be taught to exclude ovulation pain in all young women with lower abdominal pains before considering any other diagnosis.

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VAN DIE REDAKSIE

DIE ZOLLINGER-ELLISON-SINDROOM

Groot belangstelling is gewek deur die onlangse uitkenning van 'n sindroom bestaande uit hardnekkige maagverswering en grootskaalse oormatige maagsapafskeiding met verwante kliergewasse in die eilandjies van Langerhans wat nie insulien afskei nie;[1] die vernaamste kenmerke van hierdie sindroom is verlede jaar in ons briewerubriek bespreek.[2] In die kort tydjie sedert 1955 toe dit vir die eerste maal beskrywe is, het die omvang van die sindroom uitgebrei—nuwe aspekte word nou as onderdeel daarvan gereken, en by verskeie gevalle het maagverswering glad nie die siektebeeld oorheers nie. Dit is bekend dat diarree soms by hierdie siekte voorkom, maar dit gaan gewoonlik gepaard met tipiese verswering-siektes, hoewel dit ook 'n vroeë simptoom kan wees;[3] 'n geval is onlangs beskryf waarby onbeheerbare diarree ernstige kaliumtekort veroorsaak het.[4] By die lykskouing van hierdie pasiënt is 'n alvleeskliergewas ontdek wat nie onderskei kon word van die soort wat by hierdie sindroom voorkom nie, maar die enigste tekens van 'n maagsweer was bloeding uit 'n radiologies-bewese akute letsel volgend op behandeling met kortisoon.

Ook blyk dit dat ander endokrienkliere in baie gevalle aangetas kan word; Donaldson *et al.*[3] lewer 'n oorsig van 21 gevalle van hierdie sindroom en het ontdek dat gewasse of oormatige groei van die harsingslymklier, byniere of byskildklier by 6 pasiënte voorgekom het. Hoewel veelvoudige endokrien-klierwoekering 'n familiekwaal kan wees, is daar nog geen bewyse dat oorerflikheid 'n rol speel by die Zollinger-Ellison-sindroom nie. Die aantasting van die ander kliere is veelbetekenend, want by sommige gevalle is die mening uitgespreek dat die afskeidings van hierdie ander kliere aanleiding kan gee tot verswering[5] en dat die maagdermletsels van hierdie sindroom tot 'n mate daaraan te wyte kan wees. Simptome van die spysverteringstelsel kom dikwels by oormatige byskildklierwerking voor en sommige geneeshere meen dat hulle deur 'n oormaat bloedkalsium veroorsaak word, maar dit blyk dat daar bowendien 'n hoër voorkomssyfer van maagswere is;[6] daar is egter selde enigiets wat op die Zollinger-Ellison-sindroom dui by sulke gevalle. Een voorbeeld van hierdie sindroom met endokrien-klierwoekering is reeds beskrywe, waarby die tekens van oormatige byskildklierwerking die ontwikkeling van die maagswere voorafgegaan het.[5]

Hoewel hipoglisemie die afskeiding van HC1 stimuleer, bevat die eilandsélgewasse nie B-korreltjies nie en skei hulle nie insulien af nie. Dit is moeiliker om te bepaal watter rol glukagon speel; hoewel sommige werkers L-sélkorreltjies in die gewasse ontdek het[3, 7] en biochemiese bewyse dat die serum glukagon of 'n verwante stof bevat,[5] was daar geen

EDITORIAL

THE ZOLLINGER-ELLISON SYNDROME

Great interest has been aroused by the recent recognition of a syndrome consisting of intractable peptic ulceration and gross gastric hypersecretion in association with non-insulin-secreting adenomata of the islets of Langerhans;[1] its principal features were noted in our correspondence columns a year ago.[2] During the short time since it was described in 1955, the scope of the syndrome has been expanded—new components have been added, and in several instances peptic ulceration has not dominated the picture. While diarrhoea has been known to occur in this condition, it has usually been in association with typical ulcer-disease, although it may be an early symptom;[3] and now a case has been described where intractable diarrhoea produced extreme potassium depletion.[4] In this patient a pancreatic tumour indistinguishable from those described in this syndrome was found at autopsy, but the only evidence of peptic ulcer was bleeding from a radiologically-shown acute lesion which followed upon cortisone therapy.

Other endocrine glands, too, appear to be involved in many cases; Donaldson *et al.*[3] reviewed 21 cases of the syndrome and found that tumours or hyperplasia of the pituitary, adrenals or parathyroids had occured in 6. While multiple endocrine adenomatosis may be familial, there is as yet no evidence of inheritance in the Zollinger-Ellison syndrome. The involvement of the other glands has some significance, for it has been postulated in some cases that their secretions may be ulcerogenic[5] and that they may be partly to blame for the gastro-intestinal lesions of the syndrome. In hyperparathyroidism, alimentary symptoms are frequently encountered, and are thought to be produced by the hypercalcaemia, but in addition there appears to be a raised incidence of peptic ulcer;[6] however, there is rarely anything to suggest the Zollinger-Ellison syndrome in these cases. One case of this syndrome with endocrine adenomatosis has been described where the signs of hyperparathyroidism preceded the development of peptic ulceration.[5]

Although hypoglycaemia stimulates the secretion of HC1, the islet-cell tumours do not contain β-granules and do not secrete insulin. The role of glucagon is more difficult to assess; while some workers have found α-cell granules in the tumours[3,7] and biochemical evidence of the presence of glucagon or a related substance in the serum,[5] there was no

teken van oormatige afskeiding van hierdie hormoon by die oorspronklike gevalle nie,[1]

Op die oomblik kan die sindroom slegs klinies vermoed en by buikopening gediagnoseer word, en daar is gevalle waar die gewasse, selfs by operasie, nie ontdek kan word tensy die alvleesklier verwyder en oopgesny word nie. Waar daar sonder twyfel alvleeskliergewasse bestaan, moet hierdie orgaan verwyder word; dit kos 'n chirurg moed om die liggaam en stert van 'n oënskynlik gesonde alvleesklier uit te sny hopende dat hy wel gewasse daarin sal vind. By slegs een geval was dit voldoende om die neoplasma van die alvleesklier te verwyder;[4] die herhalende swere moet ook behandel word en algehele maagverwydering word aanbeveel ten tyde van die pankreatektomie.[7] Selfs al is daar uitsaaiing, word hierdie soort snykundige ingreep aanbeveel, aangesien die sekondêre neerslae swak mag afskei;[7] dit blyk dat die gewasse by ongeveer tweederdes van die gevalle kwaadaardig is en dat uitsaaiing by ongeveer die helfte gevalle ontdek is by diagnose (in die lewe of tydens lykskouing).[3]

Dit lyk onmoontlik dat hierdie vreemde siekte in verband kan staan met die gewone soort maagsweer, want by die Zollinger-Ellison-sindroom is die oormatige afskeiding van maagsappe abnormaal hoog, die swere is geneig om oor en oor te ontwikkel ten spyte van oënskynlik genoegsame snykundige ingreep, en hulle is ook geneig om op sonderlinge plekke te ontwikkel. Miskien is daar 'n anatomiese ‚eksokriene'-verduideliking van die herhaalde voorkoms van verswering in die distale dunderm of boonste jejunum: afskeidings van die eilandsélgewasse kan in die kliersakkies van die pankreas insyfer en dus *via* die hoof- en newebuise van die pankreas in die derm te lande kom.

Hierdie ingewikkelde sindroom sluit dus baie en interessante onderdele in—maagswere wat oënskynlik veroorsaak word deur hormone, neoplasie in 'n swak verstaande deel van die alvleesklier, veelvuldige endokriengewasse, en diarree. As ons eers die Zollinger-Ellison-sindroom verstaan sal 'n hele paar van ons basiese probleme opgelos word.

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evidence of hypersecretion of this hormone in the original cases.[1]

At present the syndrome can only be suspected clinically and diagnosed at laparotomy, though in some cases even at operation the tumours may not be found unless the pancreas is removed and sectioned. Where there are undoubted pancreatic tumours that organ should be removed; it takes a courageous surgeon to remove the body and tail of an apparently healthy pancreas in the hope that adenomata are present. In only one case has removal of the pancreatic neoplasm sufficed;[4] in addition, treatment of the recurrent ulcers is needed, and total gastrectomy is recommended at the same time as pancreatectomy.[7] Even where there are metastases, surgery of this type is advised, as the secondary deposits may secrete poorly;[7] it appears that the tumours are malignant in about two-thirds of the cases and that metastases have been found in half of them at the time of diagnosis (during life or at autopsy).[3]

It does not seem possible to relate this strange disease to the ordinary type of peptic ulcer, because in the Zollinger-Ellison syndrome the level of gastric hypersecretion is abnormally high, the ulcers tend to recur in spite of apparently adequate surgery, and the sites tend to be bizarre. Perhaps the frequency of ulceration in the distal duodenum or upper jejunum has an anatomical 'exocrine' explanation: secretions from the islet-cell tumours may leak into the acinar system of the pancreas and thus enter the bowel *via* the main and accessory pancreatic ducts.

Many interesting components are thus entangled in this complex syndrome—apparently hormonally-induced peptic ulcer, neoplasia of an ill-understood part of the pancreas, multiple endocrine adenomatosis, and diarrhoea. When the Zollinger-Ellison syndrome is understood, we should have answers to a number of basic problems.

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THE PROBLEM OF MIKULICZ'S DISEASE

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'I hope that future observers will succeed in solving the riddle which this remarkable disease presents to us.'

von Mikulicz, 1888.

In 1888 von Mikulicz[41] described a patient who had enlargement of the lacrimal and salivary glands. The one lacrimal gland was excised and the histology showed lymphoid infiltration with acinar atrophy.[23] The enlargement recurred and it was again excised but the patient died 2 months later from peritonitis originating in perityphlitis. This unhappy end occurred 14 months after the onset of the disease.[14, 17] What von Mikulicz described is as unknown today as it was then[11] and in view of the early death of the patient it is even impossible to say whether his patient was suffering from a benign or a malignant condition.

After that original report considerable confusion was caused by the fact that all types of cases with similar swellings were labelled as Mikulicz's disease.[16] In an attempt to restore order out of the resultant chaos, it was suggested that the term Mikulicz's syndrome should be used for those cases resembling von Mikulicz's original case but caused by some known disease[12, 20, 28, 29] such as tuberculosis, leukaemia,[13, 14]

lymphomata,[12, 21] syphilis,[14] sarcoidosis[14] and uveoparotid fever[12, 13, 36] (which is, in fact, also sarcoidosis.[14]) The diagnosis of Mikulicz's disease should then be reserved for those benign cases without any known cause.[1, 14, 28, 32, 33, 42]

THE CLINICAL PICTURE

Mikulicz's disease is a rare condition and, in a series of over 200 patients with swelling of the parotid salivary gland, one has encountered only 3 cases.

Racial Incidence. There is no information available about the racial distribution of this condition but it is possibly significant that all 3 of my cases occurred in White people, even though half of the patients with parotid enlargements were non-Whites.

Sex Incidence. All 3 patients were females. A marked predominance of females has been reported before;[8, 9, 24] its significance is as yet obscure although it naturally raises the question of some hormonal aetiology.

Age Distribution. Although there is a wide variation, this condition is particularly common in middle age,[9, 24] with an average age of 35.[33] My 3 patients were 18, 22 and 38 years of age.

The Parotid Gland. The usual history obtained is one of slowly progressive enlargement of the parotid salivary gland for weeks, months or even years.[26, 38] One or both glands may be involved,[9, 23] and the whole or only part of the gland may be affected.[8, 11, 30] At the beginning the swelling is rubbery in consistency but it becomes harder as the disease progresses.[26]

The 3 patients reported here illustrated all these classical points. The one showed involvement of a portion of the one gland only, another had enlargement of one whole gland and the third had diffuse involvement of both glands. The latter patient had been affected for 18 months only and the swellings were lobulated and rubbery in consistency, but the other two, who had been affected for 3 and 5 years respectively, had very hard glands, closely simulating a neoplasm.

Pain is usually absent and if present it is absolutely minimal.[26] This has been my experience too. Two patients had no pain at all, but the third had occasional very slight pain and 3 years after the commencement of the swelling she developed a dull, gnawing pain with occasional exacerbations at odd intervals, unrelated to meals.

The Parotid Duct, Duct Orifice and Saliva. These are always normal in this disease, but late in the disorder the affected gland will secrete very little saliva indeed. It is sometimes stated that the patients complain of a dry mouth; this should not occur, because the remaining salivary glands are still normal and should be sufficient to keep the mouth lubricated. The reported cases of a dry mouth are instances of Sjögren's syndrome, which should not be confused with Mikulicz's disease.

The Lacrimal Glands. Von Mikulicz's original case had lacrimal-gland enlargement, but as has been pointed out there is complete uncertainty about the nature of the condition he reported; it may well have been one of the conditions now grouped under the term Mikulicz's syndrome, in many of which lacrimal enlargement commonly occurs. So the fact that von Mikulicz's patient had enlargement of the lacrimal glands does not mean that lacrimal gland enlargement should be present in the disease which now carries his name.

If all cases of Mikulicz's syndrome are excluded, it is found that lacrimal enlargement is not a feature of Mikulicz's disease.[11, 26] None of the 3 patients here reported had enlargement of the lacrimal glands, although this has been seen in Mikulicz's syndrome produced by a lymphoma. It is thus difficult to evaluate reports of cases with lacrimal-gland enlargement in Mikulicz's disease,[23, 24] for one is never sure that the authors appreciate the fact that Mikulicz's disease is a separate entity.

MACROSCOPIC AND HISTOLOGICAL APPEARANCES

Macroscopic

The whole or part of the gland may be affected. The normal lobular structure is retained[24] and the increase in the fibrous tissue in the trabeculations accentuate the lobulations of the gland.

The cut surface is greyish white,[8] unlike the yellow colour of the normal gland. Occasionally an area resembling a chronic abscess is seen and in two of my patients such a cavity was encountered.

Histology

All 3 cases were examined histologically and in 1 case one was fortunate to obtain sections soon after the onset of the disease, again 1 year later, and again 3 years later, thus enabling one to follow the progress of the disease.

At the onset it seems as if there is an inflammatory response in the septa of the gland,[39] which results in an increase in the fibrous tissue of the trabeculae separating the lobules from each other (Fig. 1).

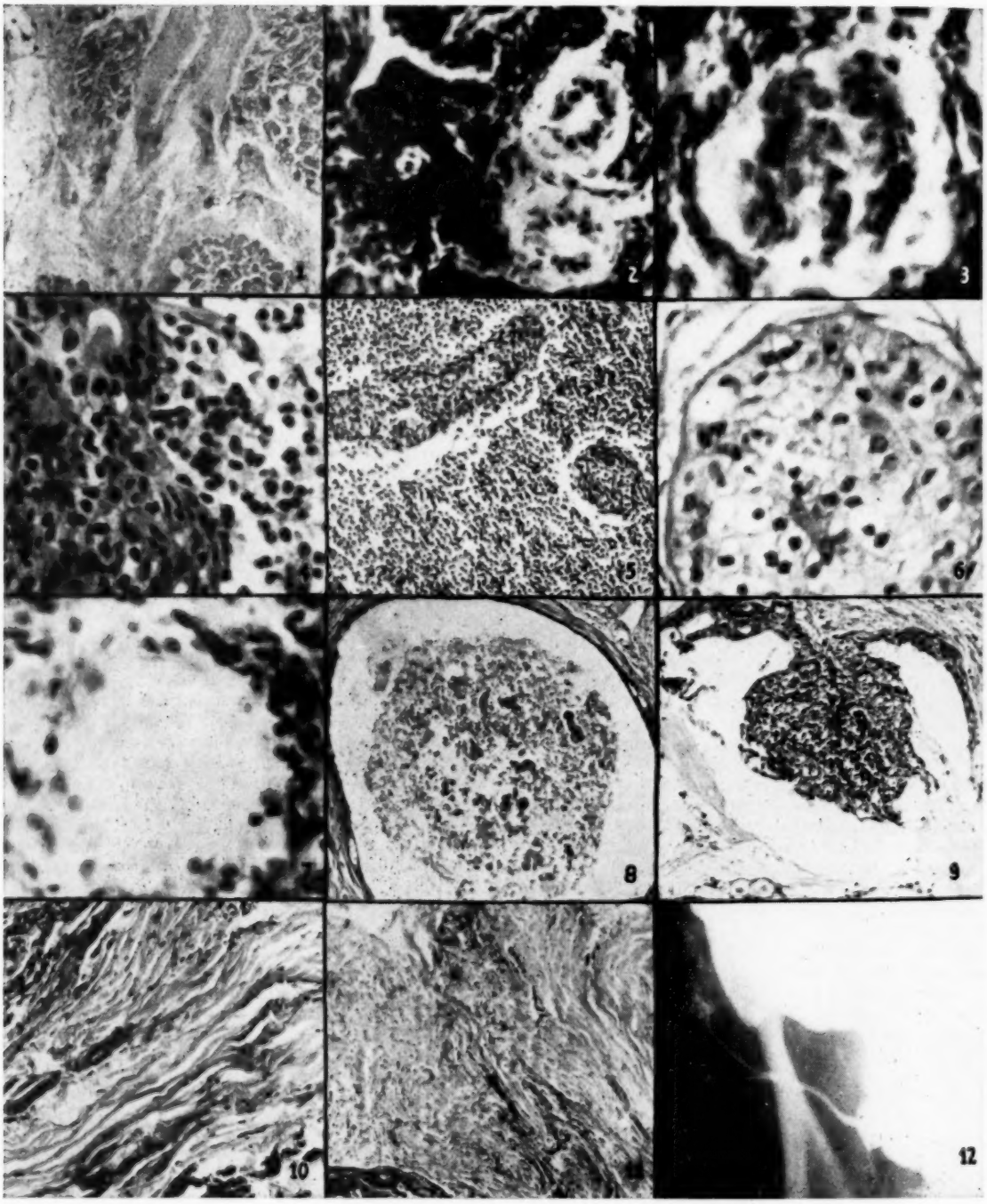
Later the process spreads to the gland parenchyma, the degree of change depending on the duration of the disease. Here the striking feature is a lymphocytic infiltration which commences around the intra-lobular ducts (Fig. 2).

The epithelium of these intra-lobular ducts soon show hyperplasia of the epithelial and myo-epithelial cells normally present in the duct;[24, 43] the epithelial cells have large ovoid, circular or reniform nuclei, while the myo-epithelial cells are thin and elongated with hyperchromatic spindle-shaped nuclei which are triangular on cross section[24] (Fig. 3). The myo-epithelial cells are also shown up by Masson's erythrosin-saffron stain, which stains the cytoplasm of myo-epithelial cells a bright pink.

At first this epithelial proliferation is confined by the basement membrane of the duct, but there is fragmentation of the reticular and collagenous tissue supporting the duct wall[26] and eventually the cells extend beyond the duct, thus resembling a malignant process (Fig. 4).

After a time the ducts become completely solid cords of cells, forming islands amongst the lymphocytes—the so-called epi-myo-epithelial islands[24] (Fig. 5 and 6). These islands eventually undergo hyaline degeneration, which increases with the duration of the condition, being minimal at 1 year, moderate at 5 years and severe after 10 years, until the whole island is replaced by an acellular acidophilic mass.[24] In one of my cases this was found to be present 3 months after the onset of the disease (Fig. 7).

This epithelial hyperplasia gradually encroaches on the lumen of the duct to produce some obstruction, with dilatation of the ducts distal to it (Fig. 8). The dilated duct may also demonstrate vigorous epithelial activity to produce small excrescences in it[24] (Fig. 9).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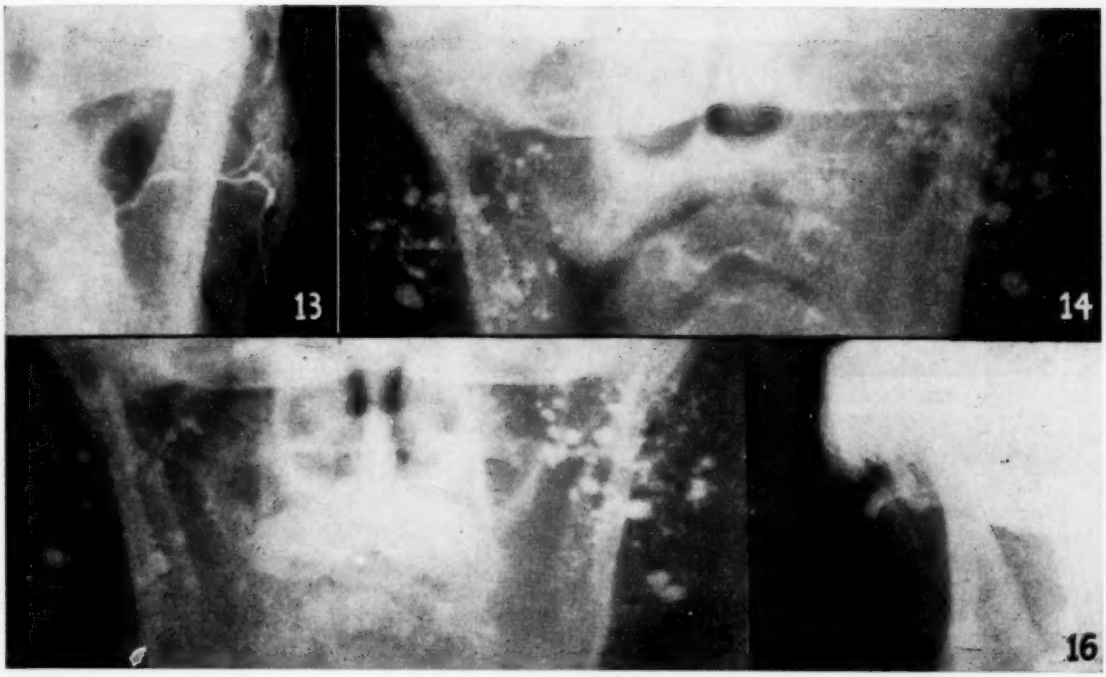
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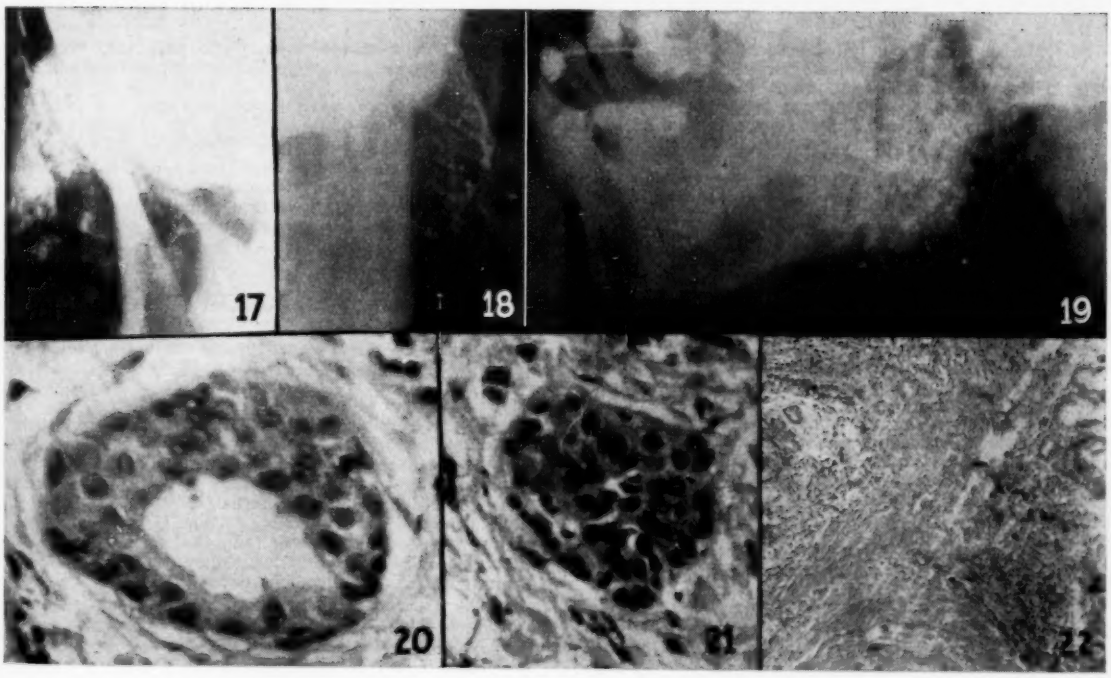
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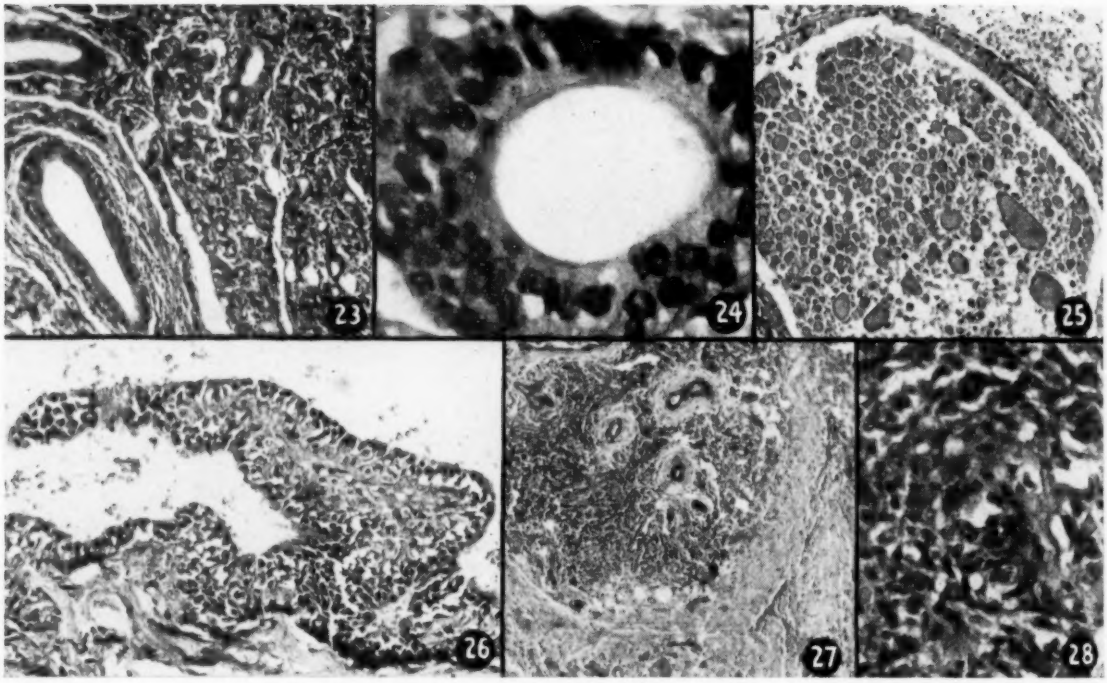

Fig. 1. Increase in fibrous trabeculations in the parotid gland 3 months after the onset of Mikulicz's disease. H. & E.

Fig. 2. Lymphocytic infiltration around the intra-lobular ducts of the parotid gland in Mikulicz's disease. H. & E.

Fig. 3. Poliferation of epithelial and myo-epithelial cells lining the intra-lobular ducts of the parotid gland in Mikulicz's disease. H. &. E.

Fig. 4. Epithelial proliferation beyond the confines of an intralobular duct of the parotid gland in Mikulicz's disease. H. & E.

Fig. 5. Epi-myo-epithelial islands in the parotid gland to Mikulicz's disease. H. & E.

Fig. 6. A higher-power view of an epi-myo-epithelial island in the same gland as in Fig. 5. H. & E.

Fig. 7. Hyaline degeneration in an epi-myo-epithelial island in the parotid gland of Mikulicz's disease. H. & E.

Fig. 8. A dilated duct in the parotid gland of Mikulicz's disease. H. & E.

Fig. 9. Epithelial proliferation into a dilated duct of parotid gland in Mikulicz's disease. H. & E.

Fig. 10. Acinar atrophy and replacement by lymphocytes and fibrous tissue in the parotid gland 15 months after the onset of Mikulicz's disease. H. & E.

Fig. 11. Marked fibrosis in parotid gland 3 years after the onset of Mikulicz's disease. H. & E.

Fig. 12. Sialogram (A.P. view) of a person with Mikulicz's disease, showing obstruction of a main intra-glandular duct.

Fig. 13. Parotid sialogram (A.P. view) in Mikulicz's disease, showing inadequate filling of the finer ductules owing to obstruction of the finer ducts.

Fig. 14. Bilateral parotid sialectasis in a case with Mikulicz's disease.

Fig. 15. Radio-opaque material still present in both glands 5 minutes after Fig. 14 during which time a lemon had been sucked for 1 minute.

Fig. 16. Sialogram taken immediately after the injection of 1·5 ml. Neo-Hydriol fluid in a patient with Mikulicz's disease.

Fig. 17. The same case as Fig. 16 but taken 24 hours later.

Fig. 18. Sialogram of an early case with typical Sjögren's syndrome (before any parotitis).

Fig. 19. Sialogram to show sialectasis in a patient with Sjögren's syndrome after many attacks of parotitis.

Fig. 20. Photomicrograph of a parotid gland after many attacks of parotitis, showing duct epithelial hyperplasia. H. & E.

Fig. 21. Photomicrograph to show a duct solid with proliferating epithelial and myo-epithelial cells in a patient with recurrent parotitis. H. & E.

Fig. 22. Acinar atrophy, lymphocytic infiltration and fibrosis in a patient with recurrent parotitis. H. & E.

Fig. 23. Periductal lymphocytic infiltration in the parotid gland of a patient with duct obstruction. H. & E.

Fig. 24. Hyperplasia of cells lining a duct in a case of parotid-duct obstruction. H. & E.

Fig. 25. A dilated duct in the parotid gland of a patient with parotid-duct obstruction. H. & E.

Fig. 26. Proliferation of epithelial cells into a dilated duct of a patient with parotid-duct obstruction. H. & E.

Fig. 27. Photomicrograph of a submandibular gland after many attacks of inflammation, showing interlobular fibrosis, acinar atrophy, and periductal lymphocytic infiltration. H. & E.

Fig. 28. Duct epithelial hyperplasia and lymphocytic infiltration in the same gland as in Fig. 27. H. & E.

While this process develops, there is a well-marked atrophy of the acini; it is uncertain why this takes place but it is tempting to postulate that it is a result of duct obstruction. As the acini disappear they are replaced by lymphocytes and fibrous tissue (Fig. 10).

The end result is a hard gland consisting almost entirely of fibrous tissue (Fig. 11), and in my own experience it is only at this stage that pain becomes a feature of the disease.

SIALOGRAPHIC APPEARANCE

As can be expected, duct obstruction with failure to fill the ducts and gland beyond the obstruction will constitute a striking feature of this condition. In one case there is obstruction of a major duct (Fig. 12), possibly due to the fibrosis and periductal lymphocytic infiltration in the septa around the inter-lobar ducts.[39] This particular patient had had a biopsy taken by her own doctor some months before and it is possible that the duct obstruction was a sequel of this operation; but it is unlikely that the biopsy was taken so deeply, and no salivary fistula followed that operation, which one would have expected if there had been damage to such a large duct.

In the second case this duct obstruction occurred in the terminal ducts, resulting in areas which failed to fill adequately (Fig. 13).

The classical sialographic appearance is that of sialectasis (Fig. 14). It has usually been assumed that sialectasis is produced by the pooling of radio-opaque material in dilated ducts, but it is in fact due to extravasation of the injected material out of the abnormally weak ductules into the interstitial tissue of the gland.[26] This view is supported by the following findings:

(i) Histologically such enormously dilated ducts are not seen.

(ii) The radio-opaque material does not leave the gland after sucking a lemon for 1 minute and taking another plate 5 minutes later (Fig. 15). In other conditions where the dye is lying in the ducts it becomes washed out by the salivary flow stimulated by sucking a lemon, as can be seen in mixed salivary tumours, in congenital sialectasis, and even in obstructions of the parotid duct.

(iii) In other parotid glands, if 1·5 ml. of dye is injected it produces acinar filling which empties very readily after sucking a lemon. In one of my patients described here, however, the injection of 1·5 ml. of dye resulted in extravasation of the injected material into the interstitial tissues of the gland and from there it spread inside the confines of the parotid fascia, as can be seen in plates taken immediately after the injection (Fig. 16), and again 24 hours after the injection (Fig. 17).

It thus seems likely that in this condition there is an abnormal weakness of the intra-glandular ducts, which rupture and allow extravasation of dye much more readily than normally.

It is obvious that the irritation produced by this extravasation of dye will aggravate the fibrosis of this disease, and sialography should thus be done very gently.

THE NATURE OF MIKULICZ'S DISEASE

It is often stated that Mikulicz's disease and Sjögren's syndrome are the same condition,[1, 24, 39] the former being merely a less highly developed variant of the latter.[23] This view is based on the fact that the sialographic and histological appearances are very similar in the two conditions. Personally I do not hold that view; I consider Sjögren's syndrome to be primarily a condition of impaired secretion of various glands, including the salivary glands. This produces all the manifestations of the so-called sicca syndrome. The resultant dry mouth and poor salivary flow produces attacks of parotitis, and it is these attacks which produce the typical radiological and histological features. This view is based on several facts:

(i) In the early stages of Sjögren's syndrome the sialogram is normal, as can be seen in Fig. 18, from a patient with the typical sicca syndrome. Sialectasis is only seen after repeated attacks of acute parotitis, as can be seen on the plates of such a patient (Fig. 19). These plates are in no way different from those seen in any other type of recurrent parotitis.

(ii) The typical histological picture of Mikulicz's disease has been reproduced experimentally by producing obstruction and by inducing infection in the gland.[3]

(iii) Early in Sjögren's syndrome there is only glandular atrophy,[5, 6] and the typical appearance histologically is the result of the recurrent attacks of parotitis.[6]

(iv) The histological features of Mikulicz's disease is also found in recurrent parotitis and in pure duct obstruction. Thus in recurrent parotitis with sialectasis one can see duct epithelial hyperplasia (Fig. 20) with the formation of solid ducts (Fig. 21) and eventually fibrosis and acinar atrophy with lymphocytic infiltration (Fig. 22). Similarly, in a case with duct obstruction there was lymphocytic infiltration (Fig. 23), duct epithelial proliferation (Fig. 24), dilated ducts (Fig. 25) with excrescences into them (Fig. 26).

It is thus obvious that this histological picture of acinar atrophy, proliferation of duct epithelium, lymphocytic infiltration and fibrosis is not pathognomonic of any one disease, and I suggest that it is a non-specific reaction on the part of the parotid to chronic irritation,[11] whatever the cause of the irritation. This same histological picture may even be seen in the submandibular salivary gland which has been subjected to recurrent attacks of infection (Figs. 27 and 28).

The diagnosis of Mikulicz's disease must thus not be made on the basis of the histological picture alone, as is so frequently done,[39] but on the combination of the clinical picture, sialography and histology. If these strict criteria are used in the diagnosis of Mikulicz's disease it will not be confused with Sjögren's syndrome and it will be found that many cases reported in the literature as cases of Mikulicz's disease are in fact some other condition, notably recurrent parotitis whether due to Sjögren's syndrome or not.

It is as yet uncertain what the cause of the chronic irritation is in Mikulicz's disease. It may be exogenous or endogenous.[2] Of the exogenous causes, infection is the one most frequently suggested,[11, 32] but no organism has ever been found in these cases and in my own cases the saliva has been sterile. In one case the contents of a cavity found in the centre of the mass at operation was also sterile on culture. In the patient with the bilateral swellings a cystic mass appeared on the one side 6 months after the biopsy and 3 months after the small dose of deep X-ray therapy. Aspiration revealed a thick fluid which was sterile on culture and consisted of necrotic material only. It had a salivary-amylase content of 100 units per ml.

All this strongly suggests that Mikulicz's disease is not due to a bacterial infection but it does not, of course, rule out a virus infection. This is now being investigated, because

there is ample evidence of viruses infecting the parotid gland in other conditions such as mumps, cytomegalic inclusion disease[44] and, more recently, in association with herpangina.[45] Dr. A. Kipps very kindly investigated the fluid aspirated from the cyst in the gland of the one patient described above. No virus was found in the fluid. This one case still does not exclude a virus as the causation, of course, more especially as this patient had received deep X-ray therapy before the fluid was aspirated.

Of the endogenous causes, it has been suggested that it may be one of the connective-tissue diseases in view of an association with disseminated lupus erythematosus;[23] but this has not been confirmed.

A most tempting suggestion is that there is an increased permeability of the intra-glandular ducts,[24] and that this allows saliva to escape into the interstitial tissue of the gland to produce the irritation.[26] This is pure hypothesis at present but it would fit in with the disease as we know it. In the hope of finding some auto-sensitivity to saliva in these people, I have investigated one patient fully, with the following findings: White blood count: 8760 per c.mm., polymorphs 62%, lymphocytes 30%, monocytes 2%, eosinophils 6%. Sedimentation rate: 1 mm. in 1 hour. Haemoglobin 15 g.%. Serum albumin 5·5 g.%, serum globulin 2·3 g.%, serum calcium 11 mg.%. Thymol turbidity 4·4. Zinc turbidity 7·4. Alkaline phosphatase 2·6 Bodansky units.

Four weeks after a course of deep X-ray therapy to both the parotid glands, with good effect, the results were: Thymol turbidity 4. Zinc turbidity 2. Serum albumin 4·4 g.%, serum globulin 4·8 g.% (i.e. an increase in total globulin). Electrophoresis showed this increase to be due to an increase in alpha 2 globulin.

Whether the eosinophilia and the increased alpha 2 globulin have any significance is not certain, but there seems little evidence of a sensitivity reaction.

A precipitin test (kindly performed by Dr. A. Kipps) between the patients' serum and a saline extract of another person's parotid gland was also negative.

TREATMENT AND PROGNOSIS

Because its nature is obscure, the treatment we can offer must be empirical.

The management of this condition is largely controlled by the suspicion or fear that it is a neoplasm, and consequently most people will insist on a biopsy before any decision is made regarding treatment.[11, 26]

If the condition is localized, it will resemble a neoplasm so closely that it would be wise to excise the mass without biopsy unless facilities for frozen sections are available. Such an excision was performed on one of the 3 cases here reported and she is well.

If it is a diffuse condition in the relatively early stages with a rubbery consistency (suggesting lymphocytic infiltration without much fibrosis) then deep X-ray therapy in small doses may be most effective.[8, 9, 11, 14, 16, 17] This was used in my one patient with bilateral parotid involvement, who was given the equivalent of 850 r total to each side with a remarkably satisfactory result.

In the later stages, when the gland is hard owing to fibrosis, parotidectomy should be performed (with preservation of the facial nerve) if the symptoms (pain or disfigurement) warrant it.[26, 38] This was done on one of my patients with a satisfactory result.

Prognosis. There is no mortality associated with this disease and the prognosis must thus be said to be good.[9] The local mass may disappear with radiotherapy; if not, parotidectomy will remove it successfully. Recurrences, however, do occur,[11] or, in some cases, the opposite side may become affected.[11]

CONCLUSIONS

Mikulicz's disease should thus be diagnosed on the basis of the clinical, sialographic and histological pictures taken together because the sialographic appearance is not pathognomonic and the histological features are those of a non-specific response to chronic irritation.

The aetiological factor responsible for this condition is as yet obscure, but two aspects are being investigated: the possibility of a virus infection and the interesting theory that it may be due to a sensitivity of the gland to some constituent in the saliva.

It remains only to consider whether we should continue calling this condition Mikulicz's disease. The name has the advantage that it is a generally known term but the objections are that it may become confused with Mikulicz's syndrome and that there is no proof that the case von Mikulicz originally described was in fact the condition we are discussing.

One thus feels that the term should be abandoned; the difficulty, however, is to find a suitable substitute. Various names have been proposed but they all have their objections; e.g. benign lympho-epithelial disease[11] (apt to be confused with lympho-epithelioma), lymphocytic tumour[18] (but it is not a tumour in the common usage of the term), chronic sialectatic parotitis[26] (but true sialectasis is not present), adenolymphoma[15, 19, 27] (but we reserve that name for a very specific condition[42]). It certainly seems like an inflammatory response to an irritant which is as yet undetermined, and it would thus seem to be logical to call the condition idiopathic chronic parotitis.

SUMMARY

Mikulicz's syndrome must be clearly distinguished from Mikulicz's disease.

The histological picture of Mikulicz's disease is acinar atrophy, periductal lymphocytic infiltration, duct-epithelium proliferation and fibrosis. This is not specific for this condition but is a non-specific response on the part of the parotid gland to any chronic irritation. It is even seen in the submandibular salivary gland after recurrent attacks of inflammation.

A better name for this condition is Idiopathic Chronic Parotitis.

The various sialographic appearances of this disease are described; none of them are pathognomonic.

This condition can thus only be diagnosed by a consideration of the clinical, sialographic and histological pictures.

The expenses of this investigation were defrayed by a grant from the Dr. C. L. Herman Research Fund. I am indebted to the clinicians who referred these patients to me, to Prof. J. H. Louw for the facilities to study the cases, Dr. A. Kipps for the precipitin test, the Superintendent of Groote Schuur Hospital for permission to publish, and Mr. G. McManus for the photographs.

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HAEMOLYTIC ANAEMIA AND ERYTHROCYTE SENSITIZATION IN THE MALIGNANT RETICULOSES

WITH A REPORT OF THREE CASES

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Anaemia not infrequently occurs in patients suffering from various types of the so-called malignant reticuloses. The cause of the anaemia, when not due to extravascular blood loss, is problematical. Up till 50 years ago it was accepted as being the result of simple mechanical replacement of the erythropoeietic tissues. This view still has many adherents.[1-6] Hirschfeld,[7] in 1906, was the first to suggest that increased destruction of erythrocytes may play a part in the pathogenesis of the anaemia. During the past 20 years the haemolytic theory has become increasingly popular, mainly as the result of numerous reports of secondary haemolytic anaemia occurring in leukaemia,[8-17] Hodgkin's disease,[11, 18-23] lymphosarcoma,[14, 16, 21, 24] reticulum-cell sarcoma,[24] reticuloendotheliosis,[17] histiocytic medullary reticulosis,[24] giant follicular lymphoblastoma,[14] and multiple myelomatosis.[23] With the development of reliable techniques, shortening of the life span of the red blood cells has been demonstrated to be a significant feature in patients suffering from the malignant reticuloses.[25-28] Seaman *et al.*[29] in analysing 212 cases of chronic lymphatic leukaemia noted the presence of overt leukaemia in 52 patients. They concluded that the development of haemolytic anaemia appeared to be a random effect developing as a function of the duration of the leukaemia, with one case of haemolytic anaemia occurring for every 231 months of leukaemic life.

The cause of the increased rate of red blood-cell destruction has been the subject of many speculations and investigations. It has been attributed to phagocytosis of erythrocytes by proliferating histiocytes,[11] to spherocytosis of erythrocytes with resultant increased fragility,[14] to increased splenic activity,[25, 30] and to the haemolytic effect of metabolites generated by proliferating lymphadenomatous or reticulosarcomatous tissue.[30] With the development of immunohaematological techniques during the last decade it has become evident that in many instances the associated haemolytic anaemia has an immunological basis, as evidenced by erythrocyte sensitization (positive direct Coombs test) and, in some cases, the demonstration of circulating anti-erythrocyte antibodies.[15, 24, 26, 30-33]

Various suggestions have been made to explain the development of 'auto-antibodies' by an individual against components of his own tissues. Proof for any of these theories is still lacking, and as yet no single hypothesis has gained universal acceptance. The disease process may in some way alter the red blood cells, causing them to become auto-antigenic, and resulting in the formation of antibodies directed specifically against erythrocytes. It is also possible that the tissue proteins in the pathological lesion may be altered to become auto-antigenic, yet retaining sufficient immunological resemblance to the original unmodified tissue protein to enable cross reactions to occur between the antibody and the unmodified protein. The erythrocyte sensitization (and the haemolytic anaemia) would then be part of a generalized autoimmune process, affecting many of the body's tissues. Such a generalized immunological process could well be the explanation of the 'toxic' effect of certain diseases. It has also been suggested that 'foreign' protein generated in the pathological tissue could combine with tissue haptene. The specificity of such complexes of foreign protein and tissue haptene would be determined by the haptene.

CASE REPORTS

Case 1

The patient was a middle-aged European male, suffering from chronic lymphatic leukaemia. When he first came under observation the haemoglobin concentration was 10·0 g. per 100 ml. There were 229,500 leucocytes per c.mm., with 2·5% neutrophil polymorphonuclear cells, 0·5% basophil polymorphonuclear cells, 9·5% prolymphocytes, 84·0% lymphocytes and 3·5% 'blast' cells. The red blood cells were normochromic, and showed anisocytosis and increased diffuse polychromasia. The reticu-

locyte count was 2·6%. Examination of an aspirated sample of sternal marrow showed a total of 395,000 nucleated cells per c.mm., with a myeloid erythroid ratio of 4·8 : 1. There was a predominance of cells of the lymphocytic series (lymphocytes 62·0%, prolymphocytes 15·6% and lymphoblasts 3·2%).

The patient was under observation for 10 months before he died. During this period he became severely anaemic and required repeated blood transfusions. The reticulocyte count fluctuated between 2·4 and 36·0%. Increased excretion of urobilin was demonstrated in the urine, and the serum bilirubin concentration was 2·7 mg. per 100 ml., of which 0·3 mg. was direct-reacting. There had been no significant extravascular blood loss.

Immuno-haematological investigations. These were carried out for the first time approximately 1 month before the patient died. The direct Coombs test was positive, and the quantitative Coombs test gave a reaction of the 'warm' antibody type (Table I). Anti-

TABLE I. THE QUANTITATIVE COOMBS REACTION (CASE 1)

Dilutions of antiglobulin serum

1 *in* 4	1 *in* 16	1 *in* 64	1 *in* 256	*Saline control*
+	+++	+++	++	—

+ denotes weak agglutination; +++ denotes strong agglutination.

erythrocyte antibodies were demonstrable in the patient's serum against ficinated cells as well as with the indirect Coombs test, the reactions being stronger at 37°C than at room temperature (Table II). The antibodies did not agglutinate trypsinized cells

TABLE II. THERMAL AMPLITUDE OF ANTIBODY (CASE 1)

(using ficinated cells; sensitization for 1 hour)

Dilutions of the patient's serum

	1 *in* 1	1 *in* 2	1 *in* 4	1 *in* 8	1 *in* 16	1 *in* 32
Room temp.	++	+	+	(++)	(+)	—
37°C	++	++	+	+	(++)	(±)

++ denotes moderately strong macroscopic agglutination; (+) denotes weak microscopic agglutination; (±) denotes doubtful agglutination.

and haemolysins and cold saline haemagglutinins could not be demonstrated. An eluate prepared from the patient's sensitized cells showed a similar reaction, antibodies being demonstrable against ficinated cells and with the indirect Coombs test (but not against trypsinized cells), and showing greater activity at 37°C than at room temperature.

Summary: A case of chronic lymphatic laukaemia with a secondary immuno-haemolytic anaemia of the 'warm' antibody type.

Case 2

The patient, a European female aged 60 years, complained of lassitude and anorexia. These symptoms had started 3 months previously, and had become progressively more severe. The significant findings on clinical examination were cervical and axillary lymphadenopathy, splenomegaly and anaemia. Lymphosarcoma was diagnosed on histological examination of a cervical lymph node.

After the patient had been in hospital for 10 days an increase in the pallor of the conjunctivae and the oral mucous membranes was noted, although she had not suffered any extravascular blood loss. Haematological investigation showed the haemoglobin concentration to be 7·4 g. per 100 ml., the PCV 25·5% and the MCHC 29·0%. There were 13,200 leucocytes per c.mm., with 71·5% neutrophil polymorphonuclear cells, 10·5% monocytes, 16·5 lymphocytes and 1·5% eosinophil polymorphonuclear cells. The red blood cells were normochromic, and showed anisocytosis, poikilocytosis and increased diffuse basophilia. The platelets appeared to be reduced in numbers. The reticulocyte count was 2·7%. Urine analysis showed an increased excretion of bile pigments, and the serum-bilirubin concentration was 1·6 mg. per 100 ml., of which 0·2 was direct-reacting.

Immuno-haematological investigation. The specimen of blood showed spontaneous auto-agglutination at room temperature. The direct Coombs test was strongly positive, and the quantitative Coombs test gave a reaction of the 'cold' antibody type (Table III). An abnormal anti-erythrocyte antibody in the patient's

TABLE III. THE QUANTITATIVE COOMBS REACTION (CASE 2)

Dilutions of antiglobulin serum

1 *in* 4	1 *in* 16	1 *in* 64	1 *in* 256	*Saline control*
+++	++	+	±	—

+++ denotes strong agglutination; + denotes weak agglutination.

serum was demonstrable against red cells suspended in saline, as well as against enzyme-treated cells. The antibody reaction was stronger at room temperature than at 37°C (Table IV). The

TABLE IV. THERMAL AMPLITUDE OF ANTIBODY (CASE 2)

Dilutions of the patient's serum

		1 *in* 1	1 *in* 2	1 *in* 4	1 *in* 8	1 *in* 16	1 *in* 32	1 *in* 64
37°C	A	(+)	—	—	—	—	—	—
	B	+	(+)	(±)	—	—	—	—
	C	++	++	+	(++)	(+)	—	—
Room Temp.	A	(+)	(±)	—	—	—	—	—
	B	—	(++)	(+)	(+)	—	—	—
	C	++	++	++	+	(++)	(+)	—
4°C	A	+	(+)	—	—	—	—	—
	B	++	(++)	(+)	(+)	(+)	—	—
	C	+++	++	++	+	+	(+)	(+)

+++ denotes strong macroscopic agglutination; (+) denotes weak microscopic agglutination; (±) denotes doubtful microscopic agglutination.
A = Cells suspended in saline. B = Trypsinized cells. C = Ficinated cells.

activity of the antibody against enzyme-treated red cells as well as cells suspended in saline was not affected by inactivation of the complement in the patient's serum. Haemolysins could not be demonstrated, and the cold saline haemagglutinin titre was within normal limits.

Treatment and progress. The patient was transfused with 1,000 ml. of packed cells, without any untoward reaction, and treated with Meticorten (30 mg. per day). The haemoglobin concentration after the transfusion was 13·0 g. per 100 ml., and the reticulocyte count 7·2%. For 3 weeks the patient required a blood transfusion every 2-3 days, by means of which the haemoglobin concentration was maintained in the region of 10 g. per 100 ml. During this period the dose of the Meticorten was gradually increased. When the dose of 80 mg. per day was reached, there was a dramatic response with a marked diminution in the severity of the haemolytic process. At this stage X-ray therapy directed to the spleen was commenced. This resulted in a noticeable reduction in the size of the spleen and the lymph nodes. The intervals between transfusions became progressively longer, until after 3 weeks of this treatment the haemoglobin concentration remained between 10 and 11 g. per 100 ml. without any further blood transfusions. Despite the virtual cessation of the haemolytic process the patient's general condition deteriorated and she died about 3 months after admission to hospital. Two days before she died normoblasts appeared in her peripheral blood for the first time.

Summary. A case of lymphosarcoma with a secondary immuno-haemolytic anaemia of the 'cold' antibody type.

Case 3

The patient, a Bantu male aged 23 years, was admitted to hospital complaining of persistant headache and generalized body pains for 14 days. On clinical examination the significant findings were anaemia, enlargement of the liver and spleen, slight cervical lymph-node enlargement, and a temperature of 102°F. The haemoglobin concentration was 8·3 g. per 100 ml., the PCV was 24% and the MCHC 34%. There were 3,200 leucocytes per c.mm., with 26% neutrophil polymorphonuclear cells, 18% monocytes, 55% lymphocytes and 1% eosinophil polymorphonuclear cells. The red blood cells were normochromic, and showed anisocytosis and increased diffuse polychromasia. The reticulocyte count was 1·8%, and there were 2 normoblasts per 001 leucocytes. The bone marrow was hyperplastic, with a myeloid-

erythroid ratio of 0·82 : 1. The serum-bilirubin concentration was 0·2 mg. per 100 ml. Four weeks after admission to hospital Hodgkin's disease was diagnosed on histological examination of a cervical lymph node.

Immuno-haematological investigation. Shortly after admission to hospital the direct Coombs test was negative. During the first week in hospital a gradual fall in the haemoglobin level occurred; this was associated with increased excretion of bile pigments in the urine. There had been no extravascular blood loss, and the patient had not received any blood transfusions. The direct Coombs test was repeated, with a positive result. The quantitative Coombs test gave a reaction of the 'warm' antibody type (Table V). Anti-erythrocyte antibodies could not be demonstrated against enzyme-treated cells or with the indirect Coombs technique, and the cold saline haemagglutinin test was negative. Haemolysins could also not be demonstrated.

TABLE V. THE QUANTITATIVE COOMBS REACTION (CASE 3)

Dilutions of antiglobulin serum				
1 *in* 4	1 *in* 16	1 *in* 64	1 *in* 256	*Saline control*
+	++	—	—	—

– denotes weak agglutination; + + denotes moderately strong agglutination.

Treatment and progress. After the diagnosis of Hodgkin's disease had been established, nitrogen mustard therapy and X-ray therapy directed to the spleen were started. The patient also received 90 mg. of Meticorten daily. Despite the maintenance of the haemoglobin concentration at approximately 8 g. per 100 ml. without blood-transfusion therapy, the patient died 7 weeks after admission to hospital.

Summary. A case of Hodgkin's disease with a secondary immuno-haemolytic anaemia of the warm antibody type.

DISCUSSION

It is interesting to note that the immuno-haematological pattern was not similar in these 3 cases. In case 2 the antibody appeared to be of the cold variety, whereas the antibody in the other 2 cases was of the warm variety, as evidenced by the prozone phenomenon exhibited by the quantitative Coombs test. Dacie[30] also found different immuno-haematological patterns in the 2 cases he investigated—one of chronic lymphatic leukaemia with an associated immuno-haemolytic anaemia of the warm antibody type, and one of reticulosarcoma with an associated immuno-haemolytic anaemia of the cold antibody type.

It should be pointed out that in case 1 the patient was only referred for immuno-haematological investigation after having received numerous blood transfusions. The immuno-haematological findings may therefore not be a true reflection of the pre-transfusion picture.

Erythrocyte Sensitization

Zoutendyk and Gear[34] carried out direct Coombs tests on the red cells of patients suffering from various diseases, including 1 case of lymphatic leukaemia and 3 of myeloid leukaemia; in 1 of the latter the direct Coombs test was positive. Rosenthal *et al.*[33] found a significant incidence of erythrocyte sensitization in patients suffering from chronic lymphatic leukaemia.

The results of direct Coombs tests carried out in 30 patients suffering from various forms of malignant reticulosis are detailed in Table VI. Although the number of cases of each of these disease entities is too small to furnish reliable conclusions, the results demonstrate a significant total incidence of erythrocyte sensitization (12 out of 30).

The significance of erythrocyte sensitization in these cases is not yet clear. Rosenthal *et al.*[33] consider a positive direct Coombs test to be indicative of a potential haemolytic anaemia. By the periodic use of this test in cases of chronic lymphatic leukaemia they consider it possible to uncover cases in which the haemolytic mechanism has already become established, but in which the haemolysis has not yet become clinically evident. In the series they studied they were able to predict, correctly, the development of haemolytic anaemia in 3 cases.

TABLE VI. ERYTHROCYTE SENSITIZATION IN VARIOUS TYPES OF MALIGNANT RETICULOSES

Disease	*Number Investigated*	*Direct Coombs Test positive*
Chronic lymphatic leukaemia ..	9	5
Chronic myeloid leukaemia ..	5	0
Acute myeloid leukaemia ..	2	1
Multiple myelomatosis	3	2
Hodgkin's disease	7	3
Lymphosarcoma	2	1
Unclassified malignant reticulosis	2	0
Total	30	12

It would appear that in these cases erythrocyte sensitization is indicative of an immunologic process, of which frank haemolytic anaemia is an extreme expression.

CONCLUSIONS

1. Frank haemolytic anaemia, very often of the immunologic type, may occur in patients suffering from various forms of malignant reticulosis. This is usually of serious import, heralding a terminal phase. The haemolytic process may be severe, requiring repeated blood transfusions. It is important to determine whether the haemolytic anaemia is of the immunologic type, for such an anaemia is usually controllable by adequate corticosteroid therapy.[35]

2. In some cases the haemolytic anaemia is the predominant feature, and the underlying disease may remain undetected until special studies such as sternal-marrow aspiration or lymph-node biopsy are undertaken. Haemolytic anaemia cannot be termed 'idiopathic' until the possibility of an underlying malignant reticulosis has been excluded.

3. As erythrocyte sensitization in a patient suffering from a malignant reticulosis may well be indicative of a potential haemolytic anaemia, it may be worth while carrying out direct Coombs tests in these patients as a routine procedure.

4. The immuno-haematological findings in cases of immuno-haemolytic anaemia complicating the various types of malignant reticulosis do not appear to follow a uniform pattern.

SUMMARY

Three cases of immuno-haemolytic anaemia complicating various forms of malignant reticulosis are presented. The significance of erythrocyte sensitization in cases not complicated by overt haemolytic anaemia is discussed.

I wish to express my gratitude to Prof. E. H. Cluver, Director of the South African Institute for Medical Research, and to Dr. A. Zoutendyk, head of the Blood Group Research and Transfusion Laboratories of this Institute, for the facilities put at my disposal to carry out these investigations.

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A CASE OF TRAUMATIC PERFORATION OF THE INTESTINE ASSOCIATED WITH INGUINAL HERNIA

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While the literature abounds with reports of ruptured bowel due to closed abdominal trauma, few cases are recorded of intestinal rupture caused by hyperextension or torsion of the trunk. There are still fewer reports of intestinal rupture due to muscular strain associated with inguinal hernia.

CASE REPORT

Mr. B.J.M., aged 28 years, was admitted to the professorial surgical unit of the Johannesburg General Hospital on 13 October 1957 at 4.30 a.m. complaining of severe abdominal pain. He stated he had drunk a large quantity of beer on the previous afternoon and evening and, while 'wrestling' with a friend at about 11.30 p.m., suddenly twisted his body to the left and immediately experienced a severe 'stitch' in his right side. This pain soon spread over his whole abdomen, particularly the lower part. The patient was quite positive that he had not received a direct blow to the abdomen. He also stated that, at the time of experiencing his 'stitch', his right inguinal hernia had suddenly become painful and increased in size. This hernia had been present for the past 3 years and had remained in his groin as a constant lump which he had never attempted to reduce. The rest of the history was non-contributory except for an appendicectomy performed in 1951.

The patient was a well-built adult male in obvious pain. The temperature was normal, the pulse rate 96 per minute and the blood pressure 120/75 mm. Hg. Except for the abdomen, the general examination was negative. The abdomen did not move on respiration and exhibited board-like rigidity down the right side. The left side was extremely tender and rebound tenderness was present. Auscultation revealed no bowel sounds and an old right 'gridiron' appendicectomy scar was noted. The left inguinal region was normal but the right side contained a large inguino-scrotal hernia with no cough impulse. The swelling was tense and exquisitely tender and no attempt was made to reduce it.

A presumptive diagnosis was made of incarcerated or strangulated right inguinal hernia with ruptured intra-abdominal viscus or possible partial reduction *en masse* of the hernia.

Operation

Under general anaesthesia, a right inguinal incision was made and, on division of the external oblique aponeurosis, a tense, inguino-scrotal sac was delivered and isolated. The sac contained turbid greenish fluid suggestive of small-bowel contents and a large piece of greater omentum attached by firm adhesions to the fundus of the sac. There was no bowel in the sac and the neck was found to barely admit the tip of the little finger beside the protruding omentum. The redundant omentum was excised and the proximal piece was ligated and returned into the abdomen. The sac was ligated and the excess amputated. A Bassini repair with catgut was performed and the wound closed around a glove-rubber drain.

The abdomen was then opened through a right lower paramedian incision. The peritoneal cavity contained fluid identical to that found in the hernial sac and about 200 c.c. was sucked out. On examination of the bowel, a small perforation of the ileum, 15 cm. from the ileo-caecal junction on the anti-mesenteric border, was discovered. The perforation was round, the edges punched out, and there was no sign of surrounding bruising or reaction of any kind. The perforation was closed with two layers of chromic catgut sutures. Further exploration revealed an absent appendix. The caecum was noted to be quite mobile and the area of perforated ileum was thought to have been lying at the back of the right internal ring. The abdomen was closed with a stab drain to the recto-vesical pouch and a drain to the paramedian wound.

The patient made an uneventful recovery.

DISCUSSION

Aird[1, 2] has drawn attention to the association of traumatic perforation of the intestine and inguinal hernia. Obviously, in many cases the abdominal trauma is sufficiently severe to cause intestinal rupture without incriminating the associated hernia but, when the trauma is trivial, the presence of the hernia may be a contributory factor. It is remarkable that in most previously reported cases the rupture has occurred in the lower ileum, usually on the anti-mesenteric border and, with one exception,[1] the injured loop has been found lying in the abdominal cavity (usually near the internal inguinal ring) and not in the hernial sac. It is also interesting to note that the condition has not been observed in women, nor in association with any external hernia other than inguinal. The hernia has usually been long standing, though not necessarily irreducible, and usually right-sided.

An ingenious theory has been propounded by Bange (quoted by Aird[1]) to explain why the presence of a hernia should render the intestine more susceptible to violence of a trivial nature. He states that 'any sudden rise of intra-abdominal pressure is invariably associated with a rise in

intra-intestinal pressure. The intestine is, however, prevented from rupture in a normal individual by the equality of the pressure within the bowel and that applied uniformly to its external surface. If, however, one loop of the bowel is applied to the abdominal orifice of a hernial sac at the time of elevation of pressure, part of its wall is unsupported and may burst'.

Despite this hypothesis, perhaps the rupture can be satisfactorily explained on the basis of mere stretching of a relatively fixed segment of bowel or localized increase in intra-abdominal pressure. The occurrence of such cases, however, suggests that the hernia is of some importance in the pathogenesis although the actual mechanism is not apparent.

With regard to the management of the case reported here, the correct procedure would probably have been laparotomy only, with hernial repair at a later date. However, considering the fitness of the patient and in view of the pre-operative diagnosis, it was probably safer to have explored the hernia first despite the fact that sepsis might have militated against the repair performed.

SUMMARY

A case of perforation of the intestine associated with inguinal hernia and due to muscular strain is presented.

Theories as to the causation of this unusual occurrence are discussed.

I wish to thank Mr. J. A. Douglas, Acting Professor of Surgery, University of the Witwatersrand, and Dr. K. F. Mills, Superintendent, Johannesburg General Hospital, for permission to publish this case and Mr. L. Stein for his helpful advice.

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ECONOMY IN A MISSION HOSPITAL *

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My choice of subject is prompted by my current interest in financial problems at the McCord Zulu Hospital. With a monthly deficit of £900 and the Province's inability to meet our request for aid in 1957, there is reason for this interest. I have recently received two reports bearing on the matter:

Dr. H., formerly on our staff, is now a staff member in a teaching hospital to the north of us in Africa, and in reporting his impressions and observations of it he wrote: 'I doubt whether one staff member in a hundred has any knowledge of the cost of a syringe, a pound of cotton wool, an ampoule of terramycin, or of any other of the things they use daily.' He went on to tell of what to him was shocking waste, and ended with the statement that in a hospital of comparable size they spent as much on drugs and surgical supplies as McCord's spent for everything, including doctors' salaries. (Doubtless he exaggerated somewhat.)

The second report was from a representative of a big drug house. He told of visiting a doctor in the country and spending part of the afternoon with him in his office, talking business from time to time as the nurse examined urines or prepared the next patient. 'No', said the doctor, 'we are not making money though we are all busy, as you can see. I am thinking of selling out and going elsewhere'. At the end of the afternoon, the Firm's representative said: 'You told me, doctor, that you are not making anything out of your practice. Now I'll tell you why. I have been listening to your prescriptions and figuring what you have actually lost on most cases you have seen today. You have no idea of the cost of your prescriptions and so you give in excess and not only what is needed. Secondly, you do not avail yourself of alternative cheaper drugs where you could use them satisfactorily. Finally, you do not charge the cost of the drugs you use.'

Our hospital constitution contains the following clause: 'The purpose of the Hospital is: (1) To give medical and surgical care to sick men, women and children, with special reference to the Bantus but not limited to them; (2) to train nurses and midwives; (3) to train interns and technicians and such other health workers as may seem expedient from time to time; and (4) to provide staff opportunities and hospital privileges for qualified and acceptable local physicians.' Thus it become a duty to see that economy is practised as a means of survival for the hospital itself and for the future of those whom it sends out as nurses and doctors to serve their own people.

The Administrator of Natal once said: 'Dr. Taylor, when you have to ask the Province for the major portion of your support, you may expect it to assume the major part of hospital control.' It seemed a fair statement. In 1951 an increased grant-in-aid brought the Provincial subsidy up to £25,000, which represented approximately 35% of our budget, most of the balance coming in as fees for non-European patients. Only this year we have had to go back, hat in hand, to say that £25,000 from the Province, representing 22% of our present budget, is not enough. We must have more to survive.

To enable you to get the picture clearly in your minds: Our cost per bed per day, which includes the cost of extensive out patient and casualty services, the training school for some 180 pupil nurses and midwives, as well as all staff salaries, etc., works out at about 20s. 6d. per day for 1955-56. Compare this figure with your own cost per bed per day for comparable services to non-Europeans.

With limited funds to provide hospitalization and out-patient services for non-Europeans throughout the country, I accept it as a moral obligation to spend as little as possible, while at the same time giving good treatment so that (1) the Government may have more to spend on African hospitalization elsewhere, and that (2) patients when they have paid the fees required of them at McCord's may still have money left for food for their families. Beyond these two cogent reasons is a third: (3) The teaching of economy to nurse and doctor trainees so that their contribution to the health needs of their people may be the greater when they shall have left McCord's. Unless these trainees go back to their communities imbued with the ideal of good treatment for relatively low fees, their patients will not be able to afford their services.

MEASURES OF ECONOMY

Except in the Transvaal, mission hospitals are expected to depend on the collection of fees to a greater or lesser extent for their support. In Natal this is especially true. Thus the economy of our mission hospitals covers: (1) The careful collection of fees, (2) the economic use of staff and materials, and (3) care in spending. Emphasis on these three aspects of hospital administration is our concern, in part for the benefit of our bank manager, who seemingly doesn't sleep well when McCord's has an overdraft, in part for the benefit of our trainees.

Collection of Fees

At McCord's this is fortunately possible because of the nearness of King Edward VIII Hospital, a fully subsidized Provincial hospital. Most adult patients come to McCord's knowing that they will be expected to pay as out-patients from 2s. 6d. for an injection of penicillin to 8s. 6d. for a full examination plus medicine

* A paper presented at the South African Medical Congress, Durban, September 1957.

for an ordinary illness, or as ward in-patients 3s. 0d. a day if Africans and 6s. 0d. a day if Indians, including all ordinary medical and nursing care, with medicines. Most of our patients have a near relative employed in Durban to assist in fee payments.

I say 'fortunately' for the charging of fees is our most potent means of preventing overcrowding. Thus it happens that we plan so that every department except the laboratory makes some direct contribution to the finances of the hospital. As yet we have not achieved the efficiency of those nursing homes where Epsom salts and aspirins by the dose are separately charged to the patients. However, at McCord's the almoners, sisters, clerks and medical officers are all, in time, made aware of the actual or assigned cost to the patient of such special items as terramycin injections, X-rays and operations.

The collection of a deposit on admission has greatly simplified collections and cut our losses. Only in emergencies do patients expect admission without an advance payment of £2-3 for Africans and £3-5 for Indians, or alternatively a 'guarantee' from an employer. Unfortunately such guarantees are often worth no more than scraps of paper. Certain departments such as X-ray, out-patients, dispensary, operating theatre, have fees designed to help carry what we call 'overhead'. The fee charged per day is quite unrealistic, but it is all that ordinary patients can pay without depriving themselves and their families of such things as food, which is equally necessary to health. We hope that the moderation of our fees may be carried on into the private practices of our non-European doctors after they leave us.

One cannot leave the subject of collections without noting the need for some system that leaves as little temptation as possible to those who handle cash. Occasionally we 'lock the door too late'. It is advisable to divide the staff into (1) those who record and charge for services, (2) those who collect these charges and issue receipts and (3) those who accept such cash for deposit; and to keep appropriate records at the different levels.

Of the fees we charge, approximates 99·4% are collected: or, put in another way, we write off £400 a year as against the £71,000 odd we collect. Discounts or cancellations marked 'to charity, are not included in that £400. It is perhaps worth noting that fees paid for services rendered amount to £71,633 a year, or 68·1% of our total expenditure. You can compare your own fee collection with this percentage.

Dr. McCord believed, and I agree, that Africans, and in fact all people, set a higher value on that for which they are charged (even though they may attempt to evade payment) than on that which they get for nothing. In charging what we feel patients can reasonably pay, we still at times are criticised by some for charging anything at all as a mission hospital. On the other hand, members of the medical profession doubtless level the same accusation at us as that levelled at Provincial hospitals of 'unfair competition with private practice'. What often happens is that McCord's is a place to go when, like the woman in the Bible 'the woman had wasted all her substance on many doctors'. So much for collections.

Economic use of Staff and Materials

This is a goal relatively unattainable but worthy of pursuit. In the employment of non-European nursing staff, our salary scales are those set by the Province, or slightly below as a mission hospital. We have more senior non-Europeans in proportion to the European staff than the Provincial Hospitals. To illustrate: Of the 9 sisters responsible for the patient's care in the hospital, assisted by non-European staff nurses, 4 are European, 5 are African. Our low ratio of trained to untrained staff is another means of saving. For example, one European sister with a part-time relief supervises the Nurses' Home with its 180 pupil nurses, midwives and resident non-European nursing staff. In passing may I say that to me 'equal pay for equal work' is a shibboleth impossible to define. The differentiation into married and single cost of living, seen in European work, denies it. But I do subscribe in principle to the same pay for medical men doing internship, junior and senior. Conceivably the non-European's expenses may be less, considering housing for his family and the education of his children, but on the other hand he will in general finish his course more in debt and with a larger number of dependents than his European counterpart. If he is to do postgraduate work and keep abreast of the profession he must not be penalized at this time.

The hospital's paid medical staff consists of 3 full-time and 1 part-time European medical officers, 2 full-time and 2 part-time non-European medical officers, and 9 non-European interns, for a daily average of 280 in-patients and 180 daily out-patients. McCord's paid out 50% of its total expenditure for salaries, as compared with 56% at Addington and 59% at Edendale.

One constantly has to set one's face against meeting needs by the mere appointment of more staff. Often what is required is a simplification and redistribution of work. Of prime importance is the development of a team spirit among staff members. This is fostered by giving them more responsibility and so more satisfaction, by giving free medical services to the staff, by making available, where possible, the hospital's facilities for buying, and by the provision of room and board at cost where desired. Recognition of meritorious service helps to build a team spirit, small social functions being held for those going on long leave or severing their connection with the hospital. Any plan for saving in time or materials is bound to fail unless it has the support of the majority of the staff.

The stewardship of materials is presented almost as a religious duty and is repeatedly stressed as a mark of good citizenship in the classroom and in the wards. It includes the use and misuse of food, water, linen, drugs, surgical supplies, etc. 'What do you need for your purpose? What do you use? What do you waste?' An oft-heard remark in the theatre is, 'You act like a specialist, which you are not, in tying that catgut in the middle', 'Don't cut ligatures with curved scissors, they cost 4 times as much to sharpen as straight ones'.

For years I have withstood the objections of matrons and sisters to allowing mothers to have their newborn babies in bed with them (3-foot-wide beds, the extra 6 inches being for the baby). Besides pleasing the mother and baby and ensuring the baby more personal attention, this practice economizes in floor space, cots and baby linen.

Much time is spent drilling doctors on the cost of drugs and the use of cheaper and equally effective substitutes (penicillin and sulphonamides generally are as effective as aureomycin and much cheaper). I must note that doctors who come to us from medical schools and Government hospitals have much to learn in this field. Another case in point is the use of Planocaine for anaesthetics at 9d. as opposed to ethyl chloride or Pentothal plus ether and oxygen costing 3s. 0d.-5s. 0d., not including the much longer time spent waiting for the anaesthetist to get the patient under. Unnecessary lights, leaking taps, the waste of cotton wool and hot water, are daily texts for sermonettes in a mission hospital. Sometimes I ask myself why I worry about these small items and then accept the expenditure of £4 a month per person on milk and meat alone. The answer of course is that unnecessary waste is an offence against a needy society while the expenditure of so much on milk and meat is part of the treatment needed in dealing with an undernourished section of the population.

African patients and staff with their different background have so much to learn as adults of what I had drilled into me as a child! 'Turn off that tap; turn off that light; finish the food on your plate or don't take such a big helping next time.' For instance, whereas in an English hospital it is the cherished privilege of the sister or staff nurse to see to the distribution of the food, at McCord's in spite of all I can do or say, this most important task both from the standpoint of economy and of the patient's appreciation and well being, is left, or handed down, to the most junior nurse as a menial task. In consequence, the food waste on the ward never fails to elevate my blood pressure.

At this point I must comment on the insistence of the Nursing Council on standards and procedures that take no account of values and basic principles as I see them. As we are a training school, our nurses practise in the wards and theatre procedures which are calculated to get an approving nod from the examiners at the end of their courses of training. These procedures have been handed down by word of mouth and text-book from post-Listerian days, quite ignoring both their value to the patient and their cost to the hospital for laundry. Recently, in the opening of an ischio-rectal abscess I counted 12 different pieces of linen being used, all of which would have to go to the laundry. By no stretch of imagination could they make the operation a sterile one. For opening a chest or knee, yes indeed, but not for the rectum or mouth. Those responsible for the training of nurses emphasize memory training and routine to the exclusion of reasoning from basic principles. As one interested in the purchase of linen and paying for its washing, this emphasis on aseptic

technique when it is basically impossible is a thing to which I object. Of course my objection is overruled by the statement, 'Yes, but what will the poor girls do when they face the examiner in their practicals?'

To get back to something more positive: Stewardship in the use of blood is a subject given too little thought. Surgeons too often order it, housemen ask for it for quite hopeless cases. It is as though we men shake our fists in the face of the merciful Angel of Death and shout 'No, you cannot bring him release from his misery and pain. With this bottle of blood I'll withstand you'. Also, there is the unnecessary routine of giving blood to patients with somewhat low haemoglobin for operations in which no severe blood loss is really expected. I deplore the ordering of blood as a routine insurance against some unpredictable disaster. If one reasons that, besides ensuring the doctor's not meeting the coroner, it will also expedite the patient's convalescence, that is another matter. Economy in the matter of blood is effected at McCord's by resisting some of the requests of the housemen for blood, calling for repeat haemoglobin examinations and at the same time getting donors to the blood bank from patients' relatives and from members of the hospital staff. We ask relatives for 2 pints of blood for each one given. Ideally, once a month the Blood Transfusion Service come up to bleed staff, nurses in training, and workers, European and non-European. As a result the blood used by McCord's does not greatly exceed the amount provided for the Transfusion Service. Incidentally this giving of blood deepens the team spirit among staff members and their feeling for the hospital's patients.

Care in Spending

This comes easily when one can neither hand on financial responsibility to those above him or alternatively cannot raise one's fees for services rendered to the public. A Provincial commission visited McCord's in 1951. After a look round and interviews with several key members of the staff such as my secretary, the bookkeeper, and the stores lady, the chairman of the commission, a leading business man and several times Mayor of Durban, asked, 'What do you pay these key people, Dr. Taylor?' His next question was, 'How do you keep them when they could command much better salaries elsewhere in the business world?' As already noted, we are fortunate in that many of our staff feel that their payment is only partly in money, that in part their payment is in the special interest they share in the work of the hospital. Thus it happens that the carpenter supervises the work of the labour pool and does excellent cabinet work, the engineer makes trolleys, tables, etc. in his spare time. For the price of 4 new wheels and 2 sheets of galvanized iron the engineer recently presented the hospital with 2 needed trolleys, thereby saving us some £30-40. Careful enquiry has shown us that the hospital can more cheaply do its own laundry than have it done outside, and can more cheaply make its own coffins for indigents than buy them outside.

And now in conclusion I recall the story of the Vermonter (in American parlance, a Vermonter comes from the same rugged background as the Scotsman, and is said to share with him many of those characteristics which we honour in our humorous prose) —this Vermonter, reflecting on things one day, reckoned that, if he could fool his cow in the matter of taste by the gradual dilution of bran with sawdust, he could in time feed her at no expense to himself from the refuse of a neighbouring saw mill. Later he regretfully told an enquiring friend that just as he had succeeded in getting the old cow off the bran entirely, she up and died of some confounded disease or other! I only hope that, in our zeal for teaching the African economy and for bringing some relief to those individuals who have to find the money that the Province spends, the hospital may not 'peter out' like the Vermonter's cow.

ADDENDA

I find I have failed to cover all the ground outlined in the précis I submitted to the Congress Committee in July 1957; hence these few addenda:

The importance of the hospital beds provided by medical missions in Natal is shown in the following compilation for the year 1956-57:

1. (*a*) The number of Europeans to each registered bed is 88 to 1. (*b*) the number of non-Europeans to each registered bed is 277 to 1.

2. The daily occupied non-European beds number 7,887. Of these, medical missions supply 43·7%.

3. There are 21 Government hospitals in Natal and 44 mission hospitals.

4. (*a*) On its maintenance of 6,090 European and non-European beds (1,854 and 4,236 respectively) the Province spends £3,858,000, or £633 per bed annually.

(*b*) On its maintenance subsidies and capital grants to mission hospitals and assisted hospitals, with their 3,051 beds (160 European and 2,891 non-European) the Province spends £242,000 or £69 per bed annually.

Answering the question, 'Is the era of mission hospitals about to end in South Africa?' my guess would be 'No'. Even if it should ultimately be deemed desirable to do away with them, there is neither personnel nor finance available to make it possible. With the Province of Natal paying only 10% to maintain a mission-hospital bed as compared with what it pays in its own hospitals, one would look rather to an extension of mission hospitalization as a measure of economy.

COMPARISON OF TWO LABORATORY METHODS FOR THE DIAGNOSIS OF PHAEOCHROMOCYTOMA

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During the past 2 years, two short laboratory screening tests for phaeochromocytoma have been published. Both methods are based on the fact that patients with a phaeochromocytoma excrete increased amounts of pressor amines (adrenaline and noradrenaline) in the urine.[1]

The normal daily output of pressor amines is 15-30 μg., but it varies quite considerably under different conditions.[2] In essential hypertension the values are usually less than 100 μg., but values as high as 180 μg. have been reported.[3] After strenuous muscular work, which represents a form of stress, there is an increase in the excretion of pressor amines.[2]

In phaeochromocytoma the excretion of pressor amines is very much increased. Usually it exceeds 250 μg. daily and it may rise to 1-3 mg.

METHODS

In the method described by Moulton and Willoughby[4] untreated urine from a patient was injected into a cat, and the effect on the animal's arterial blood pressure was observed. The response to the injected urine was compared with the response to injections of standard solutions of adrenaline and of noradrenaline (0·1-0·2 μg., per ml.). A response greater than that to the standard solutions was indicative of phaeochromocytoma. Out of 250 cases, these authors identified 7 cases of phaeochromocytoma in which the values for pressor amines ranged from 0·3-3 mg. per day.

In Hingerty's method[5] the pressor amines from the patient's urine and from a standard normal urine to which noradrenaline has been added were adsorbed onto aluminium oxide, and eluted with acid. Ferricyanide was added to develop fluorescent products, and the difference of intensity of fluorescence between standard and test under an ultra-violet lamp was compared visually. The primary object of this semi-quantitative method was to detect the presence of more than 180 μg. of pressor amines in a 24-hour urine specimen, that being the concentration in the prepared standard. Hingerty[5] applied this method to 70 cases of suspected phaeochromocytoma. In the only case in which

the diagnosis was confirmed, the daily excretion of pressor amines was about 1,500 μg. The average excretion of the other 69 cases was 46 μg. per day.

RESULTS

Since it was first published, the cat test has been applied in this department to about 50 cases of suspected phaeochromocytoma. Two positive cases were found and confirmed by operation, unfortunately before the details of the fluorescence test were published. The cat test and the fluorescence test were done in parallel on 16 cases over a period of 4 months. The results for both methods were negative, the fluorescence test giving values ranging from 20-40 μg. per day. For the purpose of comparison, artificial positives were prepared by adding noradrenaline to normal urine to give values ranging from about 150 to 500 μg. per day; the amount recoverable was approximately 30%. Frazer[6] and Riley[7] reported the same finding. A direct comparison was made by preparing a urine containing the equivalent of 400 μg. noradrenaline per day. When injected into a cat, the arterial blood pressure curve was typical of one found in phaeochromocytoma and the fluorescence test indicated a value of 130 μg. per day, showing a recovery of about 30%. In the range of 200-400 μg. per day visual comparison was very difficult. Only when the values exceeded 500 μg. was the distinction clear cut.

DISCUSSION

Both methods have advantages and disadvantages.

The cat test requires experienced operators and adequate laboratory facilities. It is a reliable method, and one advantage is the fact that up to 20 assays can be performed on the same animal. The method also allows for exact repetition of the test on the same urine sample.

The fluorescence method requires no special apparatus, but exact repetition is not possible. Furthermore a 24-hour urine specimen is recommended for this method, whereas the cat test can be performed on random samples of urine.

As far as specificity is concerned, the fluorescence method is highly specific. The adsorption on aluminium oxide is specific for dihydroxyphenyl compounds like adrenaline and noradrenaline. Monohydroxyphenyl compounds such as ephedrine and tyramine are not adsorbed.[8]

Only catecholamines with a hydroxy group on the β carbon and a hydrogen on the α carbon of the side-chain are able to form strong fluorescent compounds. Isopropylnoradrenaline (Isuprel, Aludrin) gives a strong fluorescence, while Dopamine and 3 : 4 dihydroxynorephedrine (Corbasil, Cobefrine) gives only very weak fluorescence.[9] Under the circumstances there was little reason to expect any catecholamines with a hydroxy group in the side-chain, other than adrenaline and noradrenaline, to be present in the urine.

The cat test is less specific than the fluorescence test. Two substances, histamine and ephedrine, were found to give confusing results. Histamine caused a fall in the cat's blood pressure and adrenaline sometimes had a depressor action, depending on the sensitivity of the animal. Histamine could easily be distinguished from adrenaline by the injection of antihistaminics. Ephedrine had the same action as adrenaline. It is not easy to distinguish between ephedrine and adrenaline; only by reverting to the case history was it found that one patient had been taking ephedrine for asthma.

CONCLUSION

Our experience suggests that the fluorescence test may not be sensitive enough to detect border-line cases, and is of value only when the excretion of pressor amines is very much increased.

The cat test, although less specific and more likely to be influenced by the presence of various drugs in the urine, is the more reliable test for the screening of phaeochromocytoma.

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OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

THE MEDICAL ASSOCIATION OF SOUTH AFRICA

(Incorporated in the Union of South Africa)

CIRCULAR TO MEMBERS

and

NOTICE OF AN EXTRAORDINARY GENERAL MEETING

WHEREAS it has been felt for some time past that the provisions of the Association's Regulations (i.e. Articles of Association) and By-laws with any relevant Rules lack clarity as to:

(*a*) the particular organ of the Association to conduct enquiry into allegations that the conduct of a particular Member of the Association has been detrimental to the honour and interests of the Medical Profession or Association and that he has rendered himself liable to expulsion from the Association by its Federal Council;

(*b*) the procedure which should be followed in regard to such enquiry and possible expulsion;

(*c*) the competence or power of Federal Council to make and/or adopt Rules governing the ethical and/or professional conduct of Members of the Association and as to the manner in which such power might be exercised; and

(*d*) the competence or power of Federal Council to make and/or adopt Rules of procedure governing enquiries into complaints concerning the ethical and/or professional conduct of individual Members of the Association;

AND WHEREAS with a view to achieving the necessary clarity

MEDIESE VERENIGING VAN SUID-AFRIKA

(Ingelyf in die Unie van Suid-Afrika)

OMSENDBRIEF AAN LEDE

en

KENNISGEWING VAN 'N BUITENGEWONE ALGEMENE VERGADERING

NADEMAAL daar al vir 'n tyd lank gemeen is dat die bepalings van die Reglemente (d.i. die Statute van Oprigting) en Verordeninge van die Vereniging (met bypassende Reëls) aan helderheid ontbreek omtrent:

(*a*) die besondere orgaan van die Vereniging wat ondersoek moet instel omtrent bewerings dat die gedrag van 'n besondere lid van die Vereniging skadelik is vir die eer en belange van die geneeskundige beroep of die Vereniging en dat hy homself blootgestel het aan skrapping as lid van die Vereniging deur die Federale Raad van die Vereniging;

(*b*) die prosedure wat gevolg moet word in verband met sodanige ondersoek en moontlike skrapping;

(*c*) die bevoegdheid of mag van die Federale Raad om Reëls wat die etiese en/of professionele gedrag van lede van die Vereniging beheer op te stel en/of aan te neem en die wyse waarop sodanige mag uitgeoefen kan word; en

(*d*) die bevoegdheid of mag van die Federale Raad om Reëls van prosedure op te stel en/of aan te neem wat ondersoeke van klagtes insake die etiese en/of professionele gedrag van individuele lede van die Vereniging beheer.

EN NADEMAAL sekere voorgestelde wysigings van die Statute

in the foregoing respects certain proposed amendments of the Articles of Association have been formulated;

NOW THEREFORE notice is hereby given that an extraordinary general meeting of Members of the Medical Association of South Africa will be held in the Board Room, Medical House, 35 Wale Street, Cape Town, on the second day of April 1958, at 3 o'clock in the afternoon, for the purpose of proposing for consideration and considering, and, if deemed fit, of passing with or without modification or addition in the manner required for the passing of a special resolution in terms of the Companies Act 1926, as amended, of the Union of South Africa, the following resolutions as special resolutions, namely:

That the Articles of Association of the Association be and are hereby altered, amended and added to as follows:

I. That *Article* 9 be and is hereby cancelled and deleted and that the following new Article 9 be and is hereby substituted therefore:

"9 (*a*) The Council (i.e. the Federal Council of the Association referred to and defined in Article 1) shall have power on the representation of any Division or Branch and after due enquiry by such Division or Branch either through their respective Ethical Committees or, failing such Committee in the case of any particular Division or Branch, through the relevant Executive Committee (in the case of a Division) or through the relevant Branch Council (in the case of a Branch), to expel from membership of the Association any Member of such Division or Branch (as the case may be) whose conduct shall be held by the first aforesaid Council to have been such as to render him liable to expulsion under paragraph (*d*) of the last preceding Article; provided

(i) that before passing any resolution expelling such Member the said first aforesaid Council shall have satisfied itself that the Member concerned had, by registered prepaid letter, directed to his last known address, received adequate notice of the allegations made against him and of the time and place of the enquiry held in regard thereto by the relevant Ethical Committee, Executive Committee or Branch Council, as the case may be, and further that he had been given an opportunity of defending himself before such Ethical Committee, Executive Committee or Branch Council, as the case may be;

(ii) that the Member concerned has by further registered prepaid letter directed to him as aforesaid at least 28 days before the date of the meeting of the first aforesaid Council, been notified in writing of the representation made by the said relevant Ethical Committee, Executive Committee or Branch Council, as the case may be, and has been invited to submit by registered prepaid letter directed to the Secretary of the Association, at the latest 14 days before the said date of the meeting of the first aforesaid Council, for its consideration, such counter-representation in writing in his defence as he may wish to make; and further

(iii) that in case in his aforesaid counter-representation he has made a request to that effect, he has been given an opportunity of being heard in person in his own defence by the said first aforesaid Council.

9(*b*) A majority of two-thirds of those present and voting at a meeting of the first aforesaid Council shall be required for the purpose of exercising the powers of expulsion conferred on the first aforesaid Council by the preceding paragraph of this Article;

9(*c*) Any Member who has been expelled from membership in terms of this Article shall, notwithstanding that he has ceased to be a Member, be liable to pay all sums due by him to the Association, or to any Division or Branch thereof, at the time of his expulsion;

9(*d*) No Member whose conduct is under investigation or whose conduct is the subject of enquiry as aforesaid, or in regard to whom a representation as aforesaid has been made to the first aforesaid Council shall be capable of effectively resigning his membership of the Association, nor shall his membership be terminated in pursuance of any By-law until the investigation or enquiry is completed and the decision of the first aforesaid Council is made known. An investigation or enquiry shall, for the purpose of this Article be deemed to commence at the time when the matter of such investigation or enquiry is first brought officially to the notice of the Member by the Ethical Committee or Executive Committee of the relevant Division or by the Ethical Committee or Council of the relevant Branch—as the case may be—by the direction to him of the registered prepaid letter first referred to in paragraph (*a*) of this Article."

van Oprigting met die doel om die nodige helderheid in die voormelde opsigte te verkry, geformuleer is;

SO IS DIT dat hiermee kennis gegee word dat 'n buitengewone algemene vergadering van lede van die Mediese Vereniging van Suid-Afrika in die Raadsaal, Mediese Huis, Waalstraat 35, Kaapstad, op die tweede dag van April 1958, om 3-uur in die namiddag gehou sal word, met die doel om die volgende besluite as spesiale besluite vir oorweging voor te stel en te oorweeg en, indien dit raadsaam geag word, met of sonder verandering of toevoeging aan te neem, op die wyse wat vir die aanname van 'n spesiale besluit kragtens die Maatskappywet 1926, soos gewysig, van die Unie van Suid-Afrika vereis word, naamlik:

Dat die Statute van Oprigting van die Vereniging hierby verander gewysig en uitgebrei word soos volg:

I. Dat *Artikel* 9 hierby gekanselleer en geskrap word en dat dit deur die volgende nuwe Artikel 9 vervang word:

„9(*a*). Op vertoë van enige afdeling of tak en na behoorlike ondersoek deur sodanige afdeling of tak deur òf hul onderskeie Etiese Komitees òf, waar sodanige Komitee in die geval van enige besondere afdeling of tak ontbreek, deur die betrokke Uitvoerende Komitee (in die geval van 'n afdeling) of deur die betrokke Takraad (in die geval van 'n tak), het die Raad (d.i. die Federale Raad van die Vereniging waarna verwys en wat omskrywe is in Artikel 1) die mag om enige lid van sodanige afdeling of tak (soos die geval mag wees) wie se gedrag deur laasgenoemde Raad as sodanig beskou word dat hy kragtens paragraaf (*d*) van die hieraan voorafgaande artikel geskrap kan word, finaal as lid van die Vereniging te skrap; op voorwaarde egter

(i) dat alvorens enige besluit aangeneem word waardeur sodanige lid geskrap word laasgenoemde Raad tevrede gestel is dat die betrokke lid voldoende kennis, deur aangetekende vooruitbetaalde brief na sy laaste bekende adres gerig, ontvang het van die beweringe teen hom gemaak en van die tyd en plek van die ondersoek in verband daarmee deur die betrokke Etiese Komitee, Uitvoerende Komitee of Takraad. soos die geval mag wees, en verder dat aan hom 'n geleentheid gegee was om homself voor sodanige Etiese Komitee, Uitvoerende Komitee of Takraad, soos die geval mag wees, te verdedig;

(ii) dat die betrokke lid, deur 'n verdere aangetekende vooruitbetaalde brief wat aan hom soos voormeld gerig is minstens 28 dae voor die dag van die vergadering van laasgenoemde Raad, skriftelik in kennis gestel is van die vertoë wat deur die betrokke Etiese Komitee, Uitvoerende Komitee of Takraad, soos die geval mag wees, gerig is en uitgenooi is om skriftelik, per aangetekende vooruitbetaalde brief wat ten minste 14 dae voor die voormelde datum van die vergadering van voormelde laasgenoemde Raad aan die Sekretaris van die Vereniging gerig is, sodanige teenvertoë soos hy tot sy verdediging mag verkies vir oorweging deur die Raad in te dien; en verder

(iii) dat, ingeval hy in sy voormelde teenvertoë 'n versoek tot dien effekte gedoen het, hy 'n geleentheid gekry het om persoonlik tot sy eie verdediging gehoor te word deur voormelde laasgenoemde Raad.

9(*b*) 'n Meerderheid van twee-derdes van diegene wat aanwesig is en stem op 'n vergadering van die voormelde laasgenoemde Raad word vereis vir die uitoefening van die magte van skrapping wat deur die voorafgaande artikel van hierdie Statuut aan voormelde Raad verleen is.

9(*c*) Enige lid wat in terme van hierdie artikel van sy lidmaatskap geskrap is, nieteenstaande dat hy opgehou het om lid te wees, is aanspreeklik vir die betaling van alle gelde deur hom aan die Vereniging verskuldig, of aan enige afdeling of tak daarvan, ten tyde van sy skrapping.

9(*d*) Geen lid wie se gedrag ondersoek word, of wie se gedrag die onderwerp van ondersoek soos voormeld is, of ten opsigte van wie vertoë soos voormeld tot die voormelde Raad gemaak is nie, word toegelaat om daadwerklik as lid van die Vereniging te bedank nie, nòg kan sy lidmaatskap beëindig word ingevolge enige Verordening, tot tyd en wyl ondersoek of navraag afgehandel is en die besluit van die voormelde Raad bekend gemaak is. 'n Ondersoek of navraag word vir die doel van hierdie artikel beskou om te begin op die tyd wanneer die ondersoek of navraag die eerste maal amptelik tot die kennis van die lid deur die Etiese Komitee of Uitvoerende Komitee van die betrokke afdeling, of deur die Etiese Komitee of Raad van die betrokke tak, soos die geval mag wees, gebring word deur die rigting aan hom van die aangetekende vooruitbetaalde brief waarna eerstens in paragraaf (*a*) van hierdie artikel verwys is."

II. That paragraph (*c*) of *Article* 10 be and is hereby cancelled and deleted and that the following new paragraph (*c*) be and is hereby substituted therefore:

"(*c*) Notwithstanding the provisions of the preceding sub-paragraphs (*a*) and (*b*) and without derogating from the provisions of Articles 11 and 16, no body of Members shall be finally recognised as a Division or Branch until they have complied with such conditions, if any, as regards the adoption of Rules of Organization as the Council may reasonably impose."

III. That the following new Articles numbered respectively 29*bis*, 29*tres* and 29*quat*, shall be and are hereby added to and inserted in Articles of Association immediately following the existing Article 29, namely:

"29*bis*. Without derogating from the general powers vested in the Council under Articles 28 and 29 and notwithstanding anything to the contrary contained or implied in any Regulation, By-law or Rule, for the better attainment of the objects of the Association in regard to the maintenance of the honour and interests of the Medical Profession or Association it shall in particular be competent for the Council to make and/or adopt Rules governing the ethical and/or professional conduct of individual Members of the Association by way of resolution carried by a majority of not less than two-thirds of the votes given thereon in the manner prescribed by the Regulations or By-laws—in the latter case in so far as not inconsistent with the former.

29*tres*. Complementary to the provisions of Article 29*bis* it shall also be competent for the Council to make and/or adopt Rules of procedure governing enquiries into complaints regarding the ethical and/or professional conduct of individual Members of the Association, by way of resolution passed by a simple majority of the votes given thereon in the manner prescribed by the Regulations or By-laws—in the latter case, in so far as not inconsistent with the former.

29*quat*. Insofar as there may at any time or from time to time be found to be any inconsistency or conflict between any relevant Rule of the Council as to ethical and/or professional conduct or as to the procedure to be followed in conducting any enquiry into complaints regarding ethical and/or professional conduct and any Rule which may have been passed or which may hereafter be passed by any Division or Branch, the Rule of the Council shall prevail, shall be of force and effect and shall be regarded as the Rule binding on all Divisions and Branches of the Association to the exclusion of any conflicting Rule of any Division or Branch.

IV. That *Article* 30*bis* be and is hereby cancelled and deleted.

As will be seen the effect of the foregoing suggested alterations, amendments and additions, if adopted, would be to achieve clarity in reference to the matters referred to at the commencement of this circular and notice to Members.

Notice is hereby further given in terms of Section 64 of the Companies Act, 1926—

(*a*) That if less than one-fourth of the total votes be present at the meeting, it shall stand adjourned to the same day in the following week at the same time and place.

(*b*) That at the adjourned meeting the members present in person or by proxy may deal with the business for which the original meeting was convened, and a resolution passed by not less than three-fourths of such members shall be deemed to be a special resolution notwithstanding that less than one-fourth of the total votes of the Association are represented at such adjourned meeting.

By Order of the Council,

A. H. Tonkin
Secretary

Medical House
35 Wale Street
Cape Town
28 February 1958

II. Dat paragraaf (*c*) van *Statuut* 10 hierby gekanselleer en geskrap word en dat dit deur die volgende nuwe paragraaf (*c*) vervang word:

„(*c*) Nieteenstaande die bepalings in die voorafgaande onderparagrawe (*a*) en (*b*) en sonder om af te doen van die bepalings van artikels 11 en 16, sal geen liggaam van lede finaal as 'n afdeling of tak erken word nie voordat hulle voldoen het aan sodanige voorwaardes, indien enige, ten opsigte van die aanvaarding van Reëls van organisasie soos die Raad redelik mag oplê."

III. Dat die volgende nuwe Statute respektieflik genommer 29*bis*, 29*tres* en 29*quat* hierby toegevoeg word en opgeneem word in die Statute van Oprigting onmiddellik volgende op die bestaande Statuut 29, naamlik:

„29*bis*. Sonder om af te doen aan die algemene magte wat aan die Raad toegeken is onder Statute 28 en 29 en nieteenstaande enigiets teenstrydigs vervat of behels in enige Reglement, Verordening of Reël vir die beter bereiking van die doeleindes van die Vereniging wat betref die handhawing van die eer en belange van die mediese beroep of Vereniging sal dit besonderlik in die bevoegdheid van die Raad lê om Reëls te maak en/of aan te neem wat die etiese en/of professionele gedrag van individuele lede van die Vereniging beheers deur 'n besluit wat aangeneem word deur 'n meerderheid van nie minder nie as twee-derdes van die stemme daaroor op die wyse soos deur die Reglemente of Verordeninge voorgeskryf—in die laaste geval insover dit nie in stryd is met eersgenoemde nie.

29*tres*. Aanvullend by die bepalings van Statuut 29*bis* sal die Raad ook die bevoegdheid hê om Reëls van prosedure te maak en/of aan te neem vir die ondersoek na klagtes insake die etiese en/of professionele gedrag van individuele lede van die Vereniging deur middel van 'n besluit aangeneem eenvoudig deur 'n meerderheid van die stemme daaroor op die wyse soos deur die Reglemente of Verordeninge voorgeskryf—in die laaste geval in sover dit nie in stryd is met eersgenoemde nie.

29*quat*. Insover daar te eniger tyd of van tyd tot tyd enige ongerymdheid of teenstrydigheid gevind mag word tussen enige betrokke Reël van die Raad ten opsigte van die etiese en/of professionele gedrag of ten opsigte van die prosedure wat gevolg moet word by die ondersoek van klagtes insake etiese en/of professionele gedrag en enige Reël wat miskien deur enige afdeling of tak aangeneem is of hierna aangeneem mag word, sal die Reël van die Raad geldig, van krag en in werking wees en sal beskou word as die Reël wat alle afdelings en takke van die Vereniging bind, met uitsluiting van enige strydige Reël van enige afdeling of tak."

IV. Dat artikel 30*bis* hierby gekanselleer en geskrap word.

Soos gesien sal word, sal die uitwerking van die voorafgaande voorgestelde veranderinge, amendemente en byvoegsels, indien aangeneem, wees om helderheid te kry met betrekking tot die sake waarna aan die begin van hierdie omsendbrief en kennisgewing aan lede verwys is.

Kennis word hierby verder gegee kragtens Seksie 64 van die Maatskappywet, 1926:

(*a*) Dat as op die vergadering minder as een-vierde van die totale stemme aanwesig is, sal die vergadering verdaag word tot dieselfde dag in die volgende week op dieselfde tyd en plek.

(*b*) Dat op die verdaagde vergadering die lede wat persoonlik of by volmag aanwesig is die sake waarvoor die oorspronklike vergadering belê was, mag afhandel en 'n besluit wat deur nie minder nie as drie-vierdes van sodanige lede aangeneem is as 'n spesiale besluit beskou sal word, nieteenstaande dat minder as een-vierde van die totale stemme van die Vereniging op sodanige verdaagde vergadering verteenwoordig word nie.

Op las van die Raad,

A. H. Tonkin
Sekretaris

Mediese Huis
Waalstraat 35
Kaapstad
28 Februarie 1958

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

COMPLAN

Glaxo (Laboratories) (S.A.) (Pty.) Ltd., introduce Complan and supply the following information:

Complan is to provide for the needs of patients who cannot eat a normal diet—unconscious patients, those who are unable or unwilling to swallow, after operations on the alimentary tract, plastic or maxillo-facial surgery or burns, gastrostomy patients,

ulcer cases; and for invalids, convalescents, the aged, expectant and nursing mothers, growing children, premature babies, and for special diets requiring high protein-vitamin-mineral intake. A sedentary adult, unable to eat any solid food can be fed on 1 lb. (450 g.) of Complan a day. Adults who need a high protein-vitamin-mineral supplement are usually recommended to take 4 oz. (112 g.) of Complan a day.

The composition of Complan per lb. is as follows: Protein 140 g., fat 74 g., carbohydrate 200 g., calcium 3·8 g., phosphorus 3·6 g., sodium 1·8 g., chloride (as Cl) 3·4 g., potassium 5 g., iron 36 mg., vitamin A 5,000 units, vitamin B_1 5·3 mg., riboflavine 5 mg., nicotinic acid 35 mg., pantothenic acid 13·5 mg., choline 334 mg., pyridoxine (B_6) 1·9 mg., vitamin B_{12} 10 micrograms, vitamin C 45 mg., vitamin D 1,000 units, vitamin E (acetate) 24 mg., vitamin K 5 mg., plus trace requirements.

Complan has a pleasant taste, which makes it acceptable from a cup, and being low in fat and free from starch and wheat gluten is well tolerated even by weakened digestions. In hospital it is easier to prepare a complete free-flowing tube feed or bland liquid diet with Complan than with ordinary methods. For the general practitioner it is of great value for patients nursed at home. Taken as often as necessary it forms a useful supplement when nutritional reserves are depleted.

In preparing a Complan feed, add a little cold water to the powder and whisk with a fork to a smooth cream, and then mix in the rest of the water, hot or cold as required. Plastic shakers give an excellent mixture in a few seconds, the powder here being added to the water, not *vice versa*.

As a tube feed 3 oz. (85 g.) of powder with 1 pint (568 c.c.) of water makes a 15% solution, 4 oz. (112 g.) a 20% solution, and so on. As a continuous intragastric drip 15-20% solutions are recommended. A 20% solution has a much greater buffering power than milk. Feeds may conveniently be made for 24 hours and refrigerated until required.

In a cup drink 2 oz. (57 g.) of Complan may be mixed with water and served hot, cold, chilled or sweetened; flavourings such as cocoa, coffee, and meat or yeast extracts may be added. Complan may also be added to normal food—in soups, sauces, gravies, gruels, sprinkled on cereals etc.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. C. A. R. Schulenburg, M.D., M.Ch., F.R.C.S., of Pretoria, has left for Europe, and will be visiting surgical clinics in England and on the Continent. Mr. Schulenburg will be accompanied by his wife.

* * *

Dr. H. J. H. Claassens of Cape Town, is at present working in the Department of Gynaecology at Hammersmith Hospital (Postgraduate Medical School of London), Ducane Road, Shepherds Bush, W.12.

* * *

Dr. J. L. van den Berg formerly of 7 Clarendon Avenue, Strubenvale, Springs, has started in practice as a specialist anaesthetist at 515 Sanlam Buildings, Smith Street, Durban, in partnership with Drs. Grant-Whyte, Cilliers, Silberman, Foord and Mesham. Telephone: Rooms 29725, residence 884903.

* * *

Dr. and Mrs. J. Abelsohn of Cape Town, will sail in the Carnarvon Castle on 14 March for England and America. Whilst in America Dr. Abelsohn will be the guest of Dr. Ralph Tovell in Hartford, and will visit the anaesthetic departments of hospitals in New York, Boston, Chicago, Rochester and Minneapolis.

* * *

Dr. V. M. Goetz, M.D. (Berne), of Cape Town, has left for the United States, where she will join her husband, Prof. R. H. Goetz, who has taken up an appointment as Director of Surgical Research at the Einstein College of Medicine, New York 61. Until recently Prof. R. H. Goetz was Research Associate Professor of Surgery in the University of Cape Town.

* * *

The Third Congress of the International Diabetes Federation will be held at Düsseldorf, Germany, on 21-25 July 1958. The scientific section will include the following subjects: Mechanism of the action of insulin; Reactions between the metabolism of carbohydrates and fats; Angiopathy in diabetes; Oral treatment of diabetes mellitus; The diabetic child; Care and after-care for diabetic adolescents and adults; Demography of diabetes mellitus; Driving licence and diabetes mellitus. The Banting Memorial Lecture will be delivered by Prof. Dr. G. Katsch, of Greifswald. The following discussions will be held: Recent physiological research on the metabolism of carbohydrates; Determination of insulin and insulin antibodies and inhibitors in the blood; Diabetes and pregnancy; Diabetes detection drives. The lay section will hold discussions on: Social security for the diabetic; Advice on nutrition and diet; Fight against quackery; Diet-planning and cookery demonstration; What a diabetic can do to stabilize his metabolism; Holiday camps, homes for pupils and apprentices, and employment agencies. The official Congress languages will be English, French and German, and a simultaneous language service will be in operation.

Pharmaceutical and scientific exhibitions are being planned; also a programme of entertainment and tours. Congress fee for physicians and scientists DM 45 (15 for the day), assistant doctors DM 25 (10 for the day), lay members DM 15 (5 for the day), friends DM 10. Correspondence to the Secretary, Organising Committee, Third International Diabetes Congress, II Medizinische Klinik der Med. Akademie, Moorenstrasse 5, Düsseldorf, Germany.

* * *

Unie van Suid-Afrika. Departement van Gesondheid. Aangifte van gedugte epidemiese siektes en poliomiëlitis in die Unie gedurende die tydperk: 14 Februarie 1958 tot 20 Februarie 1958.

Pes, Pokkies. Geen.

Tifuskoors. Kaapprovinsie: Malmesbury Distrik kan nou as vry van besmetting beskou word. Geen verdere gevalle is sedert die tydperk 10-16 Januarie 1958 aangemeld nie.

Poliomiëlitis

	Bl.	*Nat.*	*Kl.*	*As.*	*Totaal*
Transvaal	6	4	—	—	10
Kaapprovinsie ..	1	3	1	—	5
Oranje-Vrystaat ..	—	1	—	—	1
Natal	1	—	—	1	2
Totaal ..	8	8	1	1	18

Local Authorities

	Eur.	*non-Eur.*
Transvaal		
Carletonville Health Committee R	1	
Germiston Municipality U	1	
Johannesburg Municipality U	3	1
Meadowlands Health Committee U		1
Pilgrims Rest District R		1
Randfontein District R	1	
Roodepoort-Maraisburg Municipality U ..		1
Cape Province		
Cape Divisional Council U		1
George Municipality U		1
Kokstad Municipality U	1	
Port Elizabeth Municipality U		1
Vryburg District R		1
Orange Free State		
Winburg District R		1

Natal

Durban Borough U	..	..	..	1
Pietermaritzburg Borough U	..	..	1	

U—Urban
R—Rural

* * *

The Fifth International Congress of Internal Medicine will be held at the Sheraton Hotel, Philadelphia, USA on 23-26 April 1958 by the International Society of Internal Medicine. The previous congresses were held in Paris, London, Stockholm and Madrid. The opening meeting will be addressed by Dr. T. Grier Miller (President of the Congress), Dr. Richard A. Kern (President of the American College of Physicians) and Sir Russell Brain, Bt. (President of the Society). The Congress programme includes an impressive list of speakers from 28 countries, including the Soviet Union, Czechoslovakia, Hungary, Rumania and Poland.

In addition to lectures on special subjects, symposia and discussions will be held on: Anticoagulant therapy; Cardiac diseases; Vascular diseases; Haemotology; Cancer (medical aspects); Arthritis and rheumatism; Poliomyelitis; Other infectious diseases; Gastro-enterology; Endocrinology and diabetes; Current management of tuberculosis; Rehabilitation; Cardiovascular surgery. The addresses noted in the programme exceed 130 in number. The official languages will be English, Spanish, French and German. At most of the sessions simultaneous translations will be in operation but certain sessions will be in English only.

Social events and a post-congress tour are being arranged. The Congress will be followed by the following medical meetings in America: (1) American College of Physicians, Atlantic City, 28 April—2 May; (2) Society of Clinical Investigation, Atlantic City, 5 and 6 May; (3) World Congress of Gastroenterology, Washington, 25-29 May; (4) American Gastroenterological Association, Washington, 30 and 31 May. Enquiries regarding the Congress: Mr. E. R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pa., USA.

REVIEWS OF BOOKS : BOEKRESENSIES

PNEUMOENCEPHALOGRAPHY

Pneumoencephalography. By E. Graeme Roberston, M.D. (Melb.), F.R.C.P., F.R.A.C.P. Pp. xxi + 482. Figs. 209. £5 10s. Oxford: Blackwell Scientific Publications. 1957.

Contents: Foreword. List of Plates. I. Introductory. II. Distribution of Gas. III. A Consideration of the Mechanism of Ventricular Filling. IV. Experiments in Vitro. V. Tubular Ventricular Models. VI. Application of Principles to Practice. VII. Technique: Preliminary Considerations. VIII. Basic Encephalographic Technique and Normal Shadows. IX. Failure of Ventricular Filling: Implications and Rectification. X. The Posterior Fossa. XI. The Aqueduct of Sylvius. XII. The Third Ventricle and Neighbouring Cisterns. XIII. The Third Ventricle: Pathological Appearances. XIV. The Suprasellar Region. XV. The Lateral Ventricles. XVI. The Lateral Ventricles: Deformation by Neoplasm. XVII. The Investigation of Neoplasms of the Cerebral Hemispheres. XVIII. Difficulties in Pneumographic Diagnosis. XIX. The Subarachnoid Space Over the Cerebral Hemispheres. XX. Tomography. XXI. Atrophic Lesions: General Remarks. XXII. Vascular Diseases. XXIII. Injuries to the Head. XXIV. Infections. XXV. Hydrocephalus. XXVI. Encephalography in Childhood: A Microcosm of Encephalography. XXVII. Developmental Abnormalities. XXVIII. The Value of Pneumoencephalography. References. Index.

This book is based upon its forerunners 'Encephalography' (1946) and its successor 'Further Studies in Encephalography'. Here is considered the physics of the behaviour of gas during encephalography, especially in relation to different postural conditions and the previously published experiments *in vitro* are extended by use of glass and plastic ventricular models to simulate more closely, the natural anatomy and dynamics. These are described and excellently illustrated in Chapters IV and V, and the principles thereafter applied in practice.

These principles are argued to show that flotation of gas is insufficient to provide the motivating force for the gas to pass upwards through the aqueduct and 3rd ventricle, and to displace trapped cerebro-spinal fluid which must, in the head-flexed position, also travel upwards through the 3rd ventricle in a retrograde direction. The counter forces transmitted by vascular pulsation are thought to be unimportant, as the behaviour of the gas is similar in experimenting with the cadaver.

The possible significance of unfolding of triangular or crescentic lateral ventricles and the compressibility and elasticity of the brain, and the role played by venous volumes, are discussed.

Conclusions are drawn making analogy to a trapped gas bubble in a closed 'U' tube, and it is considered that the differential pressures inherent in the bubble, as balanced against the incompressible fluid in the limbs of the 'U' tube, causes the gas to seek its way against counter-resistance and to displace the fluid in the opposite direction, even against gravity. In the cranium the ventricles containing the gas bubble represent the one limb of the 'U' tube, and the lower ventricles and extra-ventricular spaces, the other. These experiments and fundamental deductions are ingeniously carried out and are a challenge to our casual acceptance of the phenomena of air-encephalography. They provide a basis for more logical appreciation of air techniques and interpretation which the author goes on to describe in the following chapters. In these, he focuses attention systematically upon the filling of different parts of the ventricular system, and then deals with the various pathological lesions causing ventricular distortion. In regard to the latter, this work is more comprehensive than the earlier publications dealing with neoplasms, atrophic lesions, vascular diseases, head injuries, infections, hydrocephalus and developmental anomalies.

There is a very useful chapter on encephalography in childhood, aptly qualified as 'A microcosm of encephalography'. Reference is made to tomography, the value of which is stated to be limited. The technique of the procedure is discussed and may still be thought by some, in the reviewer's opinion, to be a perfection beyond the daily scope of the practicing neurologist and even that of the neuro-surgeon and radiologist. For these practitioners, the book as a whole is perhaps the most comprehensive, while being really excellently laid out and illustrated.

To the reviewer, the outline of the physics is particularly fascinating and challenging, according in so many respects with general neuro-surgical observation. Yet one must ask whether there are not other dynamic complexities involved, such as forces set up by the phasing of rhythmic pulses in the closed cranium, pulses of large dimension from respiration, heart, abdominal compression etc., which have been ignored or only briefly considered. This criticism, if valid, in no way diminishes the value and thoughtfulness of the author's work.

K.L.A.

AORTOGRAPHY

Aortography. Its Application in Urological and some other conditions. By W. Barr Stirling, Ch.M., F.R.C.S. (Ed.), F.R.F.P.S.G. Pp. vii + 291. 155 Fig. 50s. net + Postage 2s. 1d. (Abroad). Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: I. Historical. II. Surgical Anatomy. III. Armamentarium. IV. Anaesthesia. V. Procedure. VI. Interpretation. VII. Pressure Factors. VIII. Hazards and Complications. IX. Congenital Renal Anomalies. X. Renal Cyst. XI. Renal Neoplasm. XII. Unilateral Renal Bleeding. XIII. Hydronephrosis. XIV. Renal Calculus. XV. Renal Tuberculosis. XVI. Hypertension. XVII. Renal Hypoplasia. XVIII. Post-Operative Function. XIX. Adrenal Glands. XX. Other Urological Conditions. XXI. Other Organs. XXII. Intrinsic Vascular Disease. References. Index.

The author presents a comprehensive account of a procedure of which he has had wide experience. Tribute is paid to the pioneer work of dos Santos (1929), but we are intrigued to learn that aortography was first attempted in 1896.

The chapter on Surgical Anatomy is based on aortic punctures done on cadavers and provides authoritative information not ordinarily available. In the description of technique of translumbar aortography, mention of the dos Santos needle is, surprisingly, omitted. Rubber tubing is used in preference to plastic (though the latter has the advantage of transparency) and a mechanical device is preferred to manual injection.

A chapter is devoted to hazards and complications; in the author's view the translumbar carries no greater risk than the more recent transfemoral (Seldinger) technique. We are provided with a firm basis for the interpretation of aortograms by a lucid discussion of the normal picture. The numerous excellent repro-

ductions deal mainly with renal conditions but bone tumours, splenic enlargements and peripheral arterial lesions are included, albeit briefly.

The author shows that aortography holds considerable promise in the more exact elucidation of a variety of urological conditions, especially in the ability to distinguish, in most cases, between renal cyst and tumours, which is usually impossible on clinical and pyelographic grounds alone. This may save the patient an unnecessary exploration. The information gained in conditions such as hydronephrosis, calculus, congenital anomalies etc. is of academic rather than practical value.

It seems likely though that aortography will become a *sine qua non* before nephrectomy is undertaken for a unilateral renal lesion for hypertension, and it is clearly of value in such cases of renal tuberculosis or stone where partial nephrectomy is contemplated. An unequivocal diagnosis of aneurysm of the renal artery is only possible with aortography. By clearly revealing renal function (particularly in a portion of one kidney) aortography is a distinct advanced on the accepted diagnostic procedures and will undoubtedly play an increasing role in the precise diagnosis of the more complicated urological conditions.

Mr. Stirling's book will be welcomed by Urologists, Radiologists and General Surgeons dealing with peripheral vascular disease.

P.J.M.R. and L.B.

NEUROLOGICAL DISABILITY

Ten Million and One—Neurological Disability as a National Problem. Arden House Conference Sponsored by The National Health Council. Pp. xv + 102. $3.50. New York: Paul B. Hoeber Inc. 1957.

Contents: Preface. The Authors. Acknowledgements. I. The Challenge of Neurological Disabilities Today: An Introduction to a Topic and a Conference. 2. The Economics of Neurological Disabilities. 3. Professional and Technical Management of Neurological Disabilities. 4. Preparation of the Neurologically Disabled for Mature Living—From Preschool Years Through Old Age. 5. The Outlook for Research Concerning Neurological Disabilities. 6. Who Needs to Know What About Neurological Disabilities: The Information and Education Problem. 7. Ten Million and One: A Summary of a Subject and a Conference. *Appendix A.* A Representative List of Neurological Disorders. *Appendix B.* Conference Participants Identified. *Appendix C.* Agencies Concerned With Neurological Disabilities. Index.

Good intentions, which, as everyone knows, pave the road to hell, may be so trimmed by ardent road-makers as to lose all their substance. Resolutions may be so vague as to be meaningless—the League of Nations at least managed to emphasise that point. Heaven knows, there are real and tragic neurological problems enough without adding to our difficulties by publishing a 100 page booklet which contains nothing but the muffled and almost meaningless breathings of 50 'conferees', and which ends (apart from a 30 page index) in a sentence which typifies the whole nebulosity of the book: 'We look forward to the time when we will be willing to discipline ourselves as individuals and organisations in carrying on not one but many cooperative undertakings; for we have said here that all great things achieved in community activity and in science have been the products of cooperative endeavour. Let us go forward together.'

Planning is certainly necessary in any endeavour, and the outlines of proposals are useful in concentrating efforts, but expensive volumes explaining the obvious are not to be encouraged. Open the book at random and take the first sentence: 'The importance of good diagnostic services must be stressed as well as the need for adequate treatment services.' But for whom is it necessary to stress these points? And must it be stressed at three dollars fifty for 70 pages of trite resolutions?

J.M.M.

ALGEMEENE CHIRURGIE

Lehrbuch der Chirurgie. Herausgegeben von Prof. Dr. H. Hellner, Prof. Dr. R. Nissen, und Prof. Dr. K. Vossschulte Unter Mitarbeit von M. Allgöwer, W. Baumgartner, G. Börger, H. U. Buff, H. Bürkle de la Camp, W. Dick, W. Düben, H. Ehlert, K. G. Eysholdt, E. K. Frey, H. Gelbke, G. Grundmann, R. Hasche-Klünder, G. Hegemann, H. Hellner, W. Hess, W. Hügin, J. Koncz, K. Lenggenhager, F. Loew, H. Nigst, R. Nissen, H. W. Pia, H. Poppe, L. Rathcke, P. Röttgen, M. Schwaiger, F. Stelzner, K. Tauber, K. Vossschulte, R. Wanke, E. Weisschedel, H. Willenegger. xxxvi + 1060 Seiten, 596 Abbildungen in 914 Einzeldarstellungen, Lex.-80, Ganzleinen DM 84. Stuttgart: Georg Thieme Verlag. 1957.

Inhalt: I. Entwicklung der Chirurgie. II. Wundheilung und Wund behandlung. III. Blutung und Blutersatz. IV. Chirurgische Infektionen. V. Allgemeine Geschwulstlehre. VI. Radioaktive Isotope in der Chirurgie. VII. Schock. VIII. Thrombose und Embolie. IX. Anästhesie. X. Plastiken und Transplantationen. XI. Chirurgische Technik. XII. Pathophysiologie des chirurgischen Eingriffs, Vor- und Nachbehandlung. XIII. Neurochirurgie. XIV. Vegetative Nervensystem. XV. Nebennieren. XVI. Kopf und Gesicht. XVII. Hals. XVIII. Thorax. XIX. Abdomen. XX. Urogenitalorgane. XXI. Skeletsystem. XXII. GefäBsystem und Lymphbahnen. XXIII. Grundlagen der chirurgischen Begutachtung. Sachregister.

Met enkele uitsonderings is die persone wat bygedra het tot die skrywe van hierdie handboek oor Chirurgie aan ons in Suid-Afrika onbekend. By die lees van hierdie boek besef 'n mens egter dat elke medewerker 'n meester in sy vak is—beide wat betref kennis en die manier waarop dit meegedeel word. Ongetwyfeld is van die beste chirurge in Duitsland en Switserland uitgesoek om medewerkers van hierdie handboek te wees.

Sood die inhoudsopgawe aandui, dek die boek 'n wye veld, insluitende urologie, ortopedie, neuro- en hartchirurgie. Die eerste kwart word gewy aan die basiese beginsels van chirurgie. Daarna word die verskillende dele van die liggaam behandel. Dit sal seker voldoende wees om te sê dat elke hoofstuk met welbekende Duitse deeglikheid geskryf is. Na 'n paragraaf wat kortliks die historiese agtergrond skets, volg die anatomie, fisiologie en patologie wat op daardie afdeling betrekking het. Operatiewe tegniek word nie in besonderhede gegee nie, maar die allernuutste metodes van behandeling word bespreek. So word bv. die verskillende tipes van hart-long-apparate en hul toepassing op hartkwale kortliks bespreek. Hierdie boek is vir 'n mediese handboek goed op datum. Deurgaans word klem gelê op die algemene siektetoestande, terwyl plek vir die ander in verhouding tot insidensie van voorkoms, afgestaan word. Die waarde van die boek word verhoog deur 596 foto's en tekeninge wat deurgaans goed is, en 'n bibliografie aan die einde van elke hoofstuk.

Sonder twyfel vergelyk hierdie boek goed met die allerbeste leerboeke in chirurgie in Engels. Vir die chirurg wat in 'n bepaalde afdeling spesialiseer sal dit nie veel beteken nie. Die algemene chirurg sal dit egter van groot waarde vind veral as hy op hoogte wil bly met die nuwere ontwikkelings in die gespesialiseerde afdelings van chirurgie. Vir die nagraadse student in chirurgie is hierdie 'n handboek wat die hele veld van chirurgie dek. Dit bevat egter nie vir hom genoeg besonderhede om as die enigste handboek gebruik te word nie. Die boek bereik sy hoogste waarde as handboek vir die gevorderde voorgraadse student. Vir hom dek die boek die hele veld van chirurgie in voldoende besonderhede op 'n duidelike en praktiese manier.

B.D.

CLINICAL APPLICATION OF ANTIBIOTICS

The Clinical Application of Antibiotics. Volume III. Chloramphenicol and the Tetracyclines. By M. E. Florey, M.D. Pp. ix + 303. 23 Figures. 84s. London: Oxford University Press. 1957.

Contents: Preface. *Chloramphenicol.* 1. General Considerations. 2. The Treatment of Diseases due to Specific Organisms. 3. The Treatment of Diseases considered by Systems, Age Groups or Sex. *The Tetracyclines.* 4. Chlortetracycline (Aureomycin). 5. The Treatment of Diseases due to Specific Organisms. 6. The Treatment of Diseases considered by Systems, Age Groups or Sex. 7. Effect on Growth. 8. Oxytetracycline (Terramycin). 9. The Treatment of Diseases due to Specific Organisms. 10. The Treatment of Diseases considered by Systems, Age Groups or Sex. 11. Tetracycline (Achromycin). 12. The Treatment of Diseases due to Specific Organisms. 13. The Treatment of Diseases considered by Systems. Glossary of Bacteriological Nomenclature. Bibliography. Index.

This volume, the 3rd in a proposed set of 4, deals with Chloramphenicol (chloromycetin) and the Tetracyclines (Aureomycin, Tetramycin and Achromycin). The aim of the book is to evaluate critically the data in which the present use of antibiotics is founded. The scheme of the book is to devote a separate section to each of the four antibiotics, and to treat each section under three headings, viz., General Considerations, the Treatment of Diseases due to Specific Organisms, and the Treatment of Diseases considered by systems, age or sex. There is a wealth of references, and a bibliography of forty pages. These antibiotics have been in use for a number of years, so it may not matter materially that the references go back mostly to the years before 1953. There are not many for the year 1954. Those for 1955 (the most recent) are very few indeed. (The preface is dated September 1956.)

Full as the book is of information the practicing doctor will encounter this difficulty, that although he can ascertain what the four named antibiotics can or cannot do, he will have to await the

publication of the 4th volume before he gets the advice he is looking for, viz: which antibiotic to use. The last volume will deal with Erythromycin, carbomycin and the newer antibiotics.

Complications of antibiotic therapy, such as staphylococcic enteritis and fungus infection are no more than mentioned in the third volume. This, admittedly, is owing to the overall plan of the author; but the knowledge of them and their treatment is necessary for the therapist right now.

There are many pieces of sound advice in this volume. We are advised against the temptation of administering antibiotics on inadequate data. 'The serious consequences which may follow the administration of chloramphenicol, even if rare, prompt the recommendation that it should be used only in major infections illnesses in which the specific organism has been identified as one responding better to this drug than any other available.' These are, of course, exceptions, emergencies and other desperate or urgent occasions. But certainly not as a routine measure.

These or any other antibiotics yet known have no effect on infections due to the viruses of smaller size than the psittacosis-lymphogranuloma group.

F.F.

X-RAY TECHNOLOGY

X-Ray Technology. By Charles A. Jacobi, B.Sc., R.T.(A.R.X.T.), M.T.(A.S.C.P.), M.T.(A.M.T.) and Donald E. Hagen, R.T. (A.R.X.T.). Pp. 410. 320 Illustrations. South African price 83s. St. Louis: The C. V. Mosby Company. 1957.

Contents: Chapter I. The Composition of Matter. *Chapter II.* Electricity. *Chapter III.* X-Ray Circuits. *Chapter IV.* X-Ray Physics. *Chapter V.* Preventive Maintenance. *Chapter VI.* Darkroom Technique, Equipment and Chemistry. *Chapter VII.* Ethics. *Chapter VIII.* Bones of the Extremities. *Chapter IX.* Positioning for the Extremities. *Chapter X.* Bones of the Trunk. *Chapter XI.* Positioning for the Trunk. *Chapter XII.* Bones of the Skull. *Chapter XIII.* Positioning for the Skull. *Chapter XIV.* Anatomic Physiology. *Chapter XV.* Positioning for the Viscera. *Chapter XVI.* Dental Radiography. *Chapter XVII.* Special Techniques. *Chapter XVIII.* Related Techniques. Glossary—Radiographic Terminology and Definitions.

This is an eminently practical book for the radiographer and the authors have made a valuable contribution to X-ray technology.

The chapters on matter, electricity, circuits and physics cover the subjects briefly but adequately. There are useful chapters on machine maintainance, dark-room technique and ethics, clearly written from wide experience. The authors might well have included an admonition not to leave a rotating anode running unnecessarily.

The anatomy is lucid, accurate and adequate, though an occasional diagram is too complex, e.g. fig. 33.

The greater part of the book is devoted to practical radiography. The number of positions has been kept to a minimum, which is correct in this type of book; special examinations should be directed by the radiologist as required. A few of the photographs do not illustrate the text, e.g. fig. 197 depicts a 15° not a 5° tube tilt, and fig. 201 a 30° not a 35° tilt. This is a pity as pictures are so much more easily remembered than written descriptions. Too little emphasis has been placed on the use of the erect position which most radiologists would insist on, e.g. for the sinus views. Some of the radiographs do not demonstrate the whole field of the examination, e.g. fig. 167; while others are too wide. On both radiographic and protective grounds 'coning down' should be described as 'obligatory' rather than 'desirable'.

A most useful and instructive feature that alone would make the book worth while is the tabulated routine for each examination. Radiographers have so many items to remember for each individual picture that beginners would be well advised to read this book, if only for these routines.

In spite of the few minor criticisms offered above, this volume ably achieves its author's purpose, and it can be recommended to all student radiographers and to most qualified radiographers; while the student radiologist will also find it useful. It is well arranged, illustrated and printed.

H.J.

CORRESPONDENCE : BRIEWERUBRIEK

MALE/FEMALE BIRTH RATIO OF EUROPEANS IN WEST AFRICA

To the Editor: Dr. T. Schrire[1] has raised two issues with regard to this subject. The first is that there is no scientific backing for the statement that the *y*-carrying sperm is smaller than the *x*-carrying. The second is the factor of heat in affecting spermatogenesis.

Dr. Schrire is correct in stating that there is no scientific backing for saying there is, so far as *Homo sapiens* is concerned, a difference in size between the *y*-carrying and the *x*-carrying sperm. I gave my authorities for stating that this difference existed in other species.[2] It is true that I was making deductions by analogy and that argument by analogy is always suspect. Nevertheless there appeared good grounds for advancing the theory.

Dr. Schrire suggests that the heat of the tropics may be responsible for affecting adversely the *y*-carrying spermatozoa as against the *x*-carrying one. In other words he supports my point that there is, for instance, in tropical Nigeria some factor which deleteriously affects the *y*-carrying sperms. We differ in what this factor is. He suggests it is the heat of the tropics; I have suggested quinine.

I was, of course, aware of the effect of heat on spermatogenesis but rejected the heat of the tropics as the causal factor in the male/female birth ratio of Europeans in West Africa for the following reasons.

First no scientific data exist that the testicular temperature of Europeans in the tropics is in any way raised. The normal dress for Europeans in Nigeria is a loose cotton shirt and loose cotton shorts. This is not likely to raise body temperatures or interfere with the heat-regulating mechanism of the scrotum which, owing to continuous perspiration and evaporation is kept continually moist and cool.

Furthermore, the high temperatures of the day range only from 10.30 a.m. to 4.30 p.m., that is to say over a period of 6 hours. Now both Europeans and Africans in the gold mines of South Africa do an 8-hour shift in underground temperatures consistently above these of the tropics of Nigeria, yet nothing untoward has been noticed in the birth sex ratio of miners as against the rest of the population.

The Japanese are notorious for repeatedly taking hot baths and for remaining in them for a considerable time. The human testicles then have the choice of reaching either body temperature or bath-water temperature. Either is alleged to be inimical to spermatogenesis, yet the male/female birth ratio for Japan is 1045/1000.

Furthermore, there are the Eskimo who, in their warm, furlined, windproof suits, maintain a body temperature to which the testicles must approach as there is, under such conditions, no means by which, insulated from the atmosphere and subjected to body temperature, the scrotal heat-regulating mechanism could function. There is nothing in the literature to suggest that the male/female birth ratio is in any way different for the Eskimo. The ratio for twins is reported to be the same as for Europeans.

However, the suggestion that the heat of the tropics has something to do with this difference in the male/female birth ratio falls away when further details are given. Many of the pregnancies occur in European women who have never been to the tropics. The male partner after a term of service on the coast returns by sea to Europe. The time interval, a fortnight to three weeks at sea, would restore the normal spermatogenesis in the European male. He, however, if a civil servant, had by Colonial Regulations, to continue taking his daily prophylactic dose of 5 grains of quinine during the voyage and for 3 months after landing. Banks and commercial firms followed Government practice. Pregnancies would then occur from a male system no longer exposed to heat but still exposed to quinine. Heat as a possible disturbing factor of the male/female birth ratio has been eliminated: quinine has not.

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1. Schrire, T. (1958): S. Afr. Med. J., 32, 144 (1 February).
2. Jeffreys, M.D.W. (1957): *Ibid.*, 31, 1316 (28 December).